CLINICAL ELECTROCARDIOGRAPHY

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By

DAVID SCHERF AND LINN J. BOYD
M.D., F.A.C.P. M.D., F.A.C.P.

Associate Clinical Professor of Medicine and Professor of Medicine, The New York Medical College, Flower and Fifth Avenue Hospital

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PREFACE TO THE FOURTH EDITION

In introducing this new (fourth) edition of our book it seems fitting to supplement the customary but factual statement that the text has undergone complete revision new tracings have been added and some old ones replaced by a few remarks indicating the view point adopted

The scope of the present edition like its predecessors is practical electrocardiography The physical and physiological basis of the electrocardiogram finds discussion only to the extent that it is necessary for a clear understanding of the subject While adequate reference is made to the great advances in elucidating the mechanisms involved empirical electrocardiography remains the basis for clinical recognition of abnormal patterns particularly of the T waves For this reason it has been emphasized

Without depreciating in the least the importance of great progress in many directions nor attempting to justify the special orientation of the present volume it seems timely to allude to an essential point sometimes forgotten Many explanations are tentative hypotheses which must be relinquished with the discovery of new facts Many interpretations given to different patterns in the past now are known to be wrong but the clinical value of the pattern as such remains as great as at the time of its original discovery Explanations change as knowledge of nerve and muscle physiology expands while clinical data collected on hundreds of thousands of cases each year remain an invaluable storehouse of treasure

Although this situation is not peculiar to electrocardiography in few other sciences is it more obvious In the last few years attempts have been made to minimize the value of empirical electrocardiography in contrast to rational electrocardiography In the latter every wave is explained by normal or abnormal activation of a certain area of the myocardium it is now certain that these endeavours have often led to errors Only a few years have elapsed since some enthusiastic workers stated that the standard leads were on their way out they thought unipolar limb leads were by far superior At the present time unipolar limb leads have assumed a modest place in clinical electrocardiography, and it is generally appreciated that their value in practical recognition of heart disease is limited

The particular advantage ascribed to unipolar limb leads is the fact that they reveal the potentials registered at a single area of the body rather than sums of potentials as standard leads do At the moment the pendulum is swinging in the other direction vectorcardiography has been revived "as the wave of the future" in spite of the fact that it is provided by the summation of potentials of at least two leads We have included a new section of spatial vectorcardiography because this method of analysis of the cardiac potential seems to have definite didactic value and serves certain clinical purposes We gratefully acknowledge the participation of Dr A I Schaffer who collaborated in summarizing many points (some scattered through the text) into one section for the convenience of the reader Standard electrocardiography retains however its *full* importance Entirely apart from the fact that it permits the diagnosis of arrhythmias and of certain abnormal conditions like the Pre excitation syndrome T wave changes and some small infarctions better than spatial vectorcardiography the machines employed in this procedure are at the present time too expensive for general use in routine medicine

Another contrasting situation deserves mention in this place Electrocardiography is not difficult once its many rules and laws are known With modern recorders tracings are secured in a few minutes after short training Owing to their great clinical importance electrocardiograms are obtained on practically every patient admitted to the hospital and, often on patients seen in the office or home An unwarranted notion of extreme simplicity of electrocardiography generally prevails in practice, however often it is very difficult to apply the rules and laws correctly to specific cases Consequently we have not limited ourselves to mere statement of these rules but emphasized problems created by their application For this reason the present volume is not as elementary as some books written primarily for beginners we hope it will be considered comprehensive because mistakes in diagnosis may be serious in this field

In accordance with the programme of writing a practical semi advanced textbook it seemed proper to include some uncommon phenomena to accomplish a dual purpose Apart from making the text more complete this procedure permitted the introduction of considerable physiological data Long experience has taught us that the thorough comprehension of the physiology of rare disorders often greatly facilitates understanding common disturbances In addition we have ventured to include some incompletely solved problems in

the hope of stimulating further investigations Other equally important and interesting problems the connection of contraction to the electrocardiogram for example have been intentionally omitted since a discussion of conflicting views would serve largely to confuse the reader On the other hand we have not hesitated to introduce considerable clinical material including therapy hoping thereby to bring electrocardiography even closer to clinical medicine

The impropriety of making anatomic diagnosis from the electrocardiogram remains a common sin committed against the method While the electrocardiogram greatly assists in the detection of abnormal foci in the myocardium it can say little or nothing about the nature of these foci A single tracing often does not permit to distinguish between a new or an old lesion and there are no typical waves of coronary thrombosis or coronary insufficiency or anoxia Moreover great care must be exercised in drawing conclusions from the electrocardiogram alone Not every inverted T wave has the same meaning! The finding cannot be evaluated until the interpreter knows whether the patient has received digitalis whether hypertension is present or absent and so forth Although these warnings were urged more than a decade ago in the first edition reiteration still seems indicated

The section on arrhythmias has required fewer revisions than some other parts although we have expanded our discussion of the development of extrasystoles and fibrillation The groundwork laid in this field at the beginning of the century still stands intact Some new patterns observed in special forms of infarction have been added and allusion made to myocardial participation in a number of diseases often considered to spare this structure The exercise test which one of us introduced more than two decades ago appears to attract increasing attention and finds extensive discussion Our continued interest in the T wave alterations as the result of extracardiac diseases is reflected in the text

The revision has also included changes in the bibliographies which are not intended to be exhaustive but they should provide a suitable approach to the literature on the subject

Finally this opportunity will be taken to thank many of our readers for their kind comments and to express again our appreciation to William Heinemann (Medical Books) Limited for their complete co operation

DAVID SCHERF
LINN J BOYD

September 1953

PREFACE TO THE THIRD EDITION

For this new edition (the third English edition) the entire text has been revised and many new figures added. The chest leads are discussed in greater detail and their importance for the diagnosis of myocardial infarction, bundle branch block and cardiac hypertrophy is emphasized in the appropriate sections.

A short discussion of the ventricular gradient, the œsophageal leads and the unipolar limb leads concludes the first part of the book. For the diagnosis of certain types of a posterior wall infarction the value of the unipolar limb leads seems established. For other lesions they furnish confirmatory data obtained by the standard and chest leads but rarely are decisive for the diagnosis. They have, however, the additional advantage of being more readily explained than the standard leads: on the basis of our present conception of depolarization and repolarization of the heart the form of electrocardiograms obtained by unipolar leads is easily comprehended.

Since the manuscript of this book was submitted for publication a few studies were reported in which the electrocardiogram was obtained from the inside of the right auricle and right ventricle after catheterization of the heart. This new lead has only theoretical interest.

In the section on arrhythmias fewer additions and changes were necessary. In this respect the edifice erected in the first third of this century still stands intact and only little patchwork has been necessary.

We take this opportunity to thank many of our readers for their kind comments and to our publishers William Heinemann (Medical Books) Limited, for preparing a new edition in the face of existing difficulties.

DAVID SCHERF
LINN J. BOYD

February 1948

PREFACE TO THE SECOND EDITION

THE favourable reception of this book at home as well as abroad and the publisher s request for a new edition despite prevailing world conditions has given us considerable satisfaction Although only a short time has elapsed since the first edition was reprinted we employed this interval for the complete revision of some sections the improvement and supplementation of others the replacement of some tracings and the addition of numerous new ones The general arrangement and character of the book remain unchanged In the absence of any fundamentally new discoveries during the last two years drastic alterations were unnecessary but the new edition permits the inclusion of numerous advances which have given further insight into several debatable problems

Moreover discussion of some uncommon phenomena such as super normal phase and reciprocal rhythm have been added in the second edition This decision was not easily reached since the inclusion of parasystole evoked some criticism in otherwise complimentary reviews of the first edition However we believe that the reader learns a good deal of physiology in the discussion of these rare disturbances and this facilitates the understanding of the common disorders Moreover readers may be stimulated to seek for these uncommon disturbances and experience shows that our knowledge is definitely promoted by an analysis of additional variants of these phenomena We also hold the opinion that a scientific book ought not to limit itself to alterations encountered daily Finally parasystole is observed on the average once in every 1 200 records and its presence is significant since it has not as yet been observed in a healthy individual

We are indebted to the publisher Wilhelm Engelmann for permission to reproduce Fig 36 and we are particularly grateful to our publisher William Heinemann for continued help and co operation

DAVID SCHERF
LINN J BOYD

NEW YORK
1944

PREFACE TO THE FIRST EDITION

A SERIES of excellent books on electrocardiography already exists, and the responsibility of adding to the voluminous literature on this subject is great Therefore the authors consented to undertake this work only after considerable hesitation

A knowledge of electrocardiography presupposes an acquaintance with a large number of rules and laws The acquisition of these facts requires the expenditure of considerable time and effort, but otherwise is not difficult These general facts have been adequately presented in most text books on electrocardiography Owing to the novelty of the subject and the necessity for presenting the fundamental features of electrocardiography to readers unacquainted with this field writers have very properly limited themselves to essentials Consequently an unwarranted notion of the extreme simplicity of electrocardiography has developed among those whose contact with tracings is infrequent For this reason it seems timely to present not only the usual rules but also to emphasize some of the difficulties encountered in endeavours to apply them Since these features are introduced this book is not designed primarily for those beginning their study of this field Moreover a departure has been made from similar works by the introduction of considerable clinical and therapeutic material It is hoped that this device will create a closer alliance between electrocardiography and the clinic While the limitations of the electrocardiogram find emphasis their alliance may extend the usefulness of this method In several fields of electrocardiography the widespread acceptance of some theories has unintentionally led to the notion that these useful working hypotheses have been established as true At times we have ventured to present incompletely solved aspects of these situations with the hope of stimulating further investigation

Fortunately the cost of the apparatus is no longer prohibitive and the registration of the electrocardiogram has become greatly simplified This is reflected in the rapid increase in the number of physicians who employ the electrocardiograph This situation is not entirely devoid of danger Considerable practice and patience is necessary before one can recognize the alterations, at times very slight, in the pattern of the electrocardiogram as something definitely pathological and can distinguish them from the normal picture with its innumerable variations Time and patience are not at the

disposal of everyone Not rarely an interpretation is reported without the necessary knowledge often the individual findings are regarded dogmatically and the heart is stated to be healthy or affected with great positiveness The difficulties or uncertainties associated with electrocardiographic interpretations often are not adequately stressed If the physician who receives such reports is unacquainted with these limitations he may be induced to institute or fail to institute measures which may vitally affect the fate of the patient

This circumstance alone constitutes an important reason for physicians who are not particularly concerned with electrocardiography to know its basic rules so that they may evaluate the findings reported to them In this connection it should not be forgotten that despite the study of countless tracings by innumerable observers many phases of electrocardiography are still in the formative stage The exact criteria in borderline cases for example the height and width of individual waves remain unsolved and important problems

For a long time students of electrocardiography were almost exclusively occupied with the arrhythmias which were for the most part known prior to the introduction of electrocardiography In recent years so many new and valuable advances have occurred in the electrocardiographic study of myocardial diseases that the apparatus has become indispensable in the examination of these common problems The present knowledge of the alarming incidence of myocarditis the early diagnosis of coronary artery disease the possibility of recognizing atypical cases of coronary thrombosis would have been impossible without electrocardiography Physiology and anatomy as well as the clinic have been enriched by numerous advances of fundamental importance

Although electrocardiography is frequently and widely employed it deserves more extensive use It *alone* may enable the physician to recognize the participation of the heart in diabetes diphtheria tonsillitis and in many other *conditions* The electrocardiogram alone may reveal the existence of coronary artery disease in patients who complain simply of abdominal fullness and flatulence in the absence of cardiac *symptoms* The discovery of electrocardiographic changes after exercise indicative of profound disturbance of myocardial blood supply and necessitating the diagnosis of extreme coronary stenosis in patients with vague substernal distress and clinical signs of a minimal aortitis is we believe another important advance suggestive of the great possibilities of electrocardiography

Electrocardiography is still a young science It was discovered and developed within the memory of many now living Every year witnesses an abundance of new contributions Nevertheless many fundamental problems are still unsolved, others which seemed satisfactorily clarified in the recent past have become dubious in the light of new investigations which have revealed unsuspected complexity and the necessity for further study An excellent example of this situation is provided by bundle branch block, which seemed to be a closed and minor issue The excellent work recently performed in this field has opened very intriguing vistas This situation conspires to make comprehensive presentation obsolete at an early age It also hampers authors since contrasting opinions on important and unsettled problems render their task difficult and tend to confuse the reader

For this reason it has seemed best to us to omit some important and interesting questions such as the problem of excitation, the connection between contraction and the electrocardiogram On the other hand heightened interest may be derived from a very brief discussion of the development of extrasystoles of fibrillation, and of conduction disturbances Those who wish to delve more deeply into these problems will find suggestive bibliographies appended to various sections of the text These references do not pretend to be exhaustive, in general publications reflecting the present status of our knowledge and containing additional bibliographies have been given preference

Emphasis should be placed upon one important rule of electrocardiography which is frequently violated namely that great care should be exercised in drawing conclusions from the electrocardiogram alone, without knowledge of the patient Not every inverted T wave in Lead I has the same meaning! This finding cannot be evaluated until the interpreter knows whether or not the patient has received digitalis whether hypertension is present or absent etc

It is also improper to make *anatomical* diagnoses from the electrocardiogram Under ordinary circumstances the electrocardiogram assists in establishing the presence of foci in some area of the myocardium, it does not inform the observer concerning the nature of these foci There are no typical coronary waves no electrocardiograms typical of coronary thrombosis of hypertrophy, of coronary insufficiency of anoxia etc

The recent discovery that the T waves may become altered by diseases of other organs (for example of the lungs gall bladder) since vago vagal reflexes influence the blood supply of the heart

and the fact that quiet standing can provoke abnormal electro cardiograms opens new possibilities It is possible that at times the severe electrocardiographic alterations or the heart failure in pneumonia the rapid death after aspiration of foreign bodies will be traced to the participation of similar reflexes

The problem of nomenclature also presents considerable difficulties since entirely different names are employed in many places An international agreement is urgently necessary

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DAVID SCHERF
LINN J BOYD

NEW YORK

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CLINICAL ELECTROCARDIOGRAPHY

I

PHYSICAL BASIS OF THE NORMAL ELECTRO-CARDIOGRAM AND THE NORMAL LEADS

POLARIZATION DEPOLARIZATION AND REPOLARIZATION

THE widely accepted membrane theory maintains that every resting muscle or nerve fibre is polarized The cells are considered to be surrounded by a virtual or actual semi permeable membrane which permits the positive ions (cations) to migrate outward the larger negative ions (anions) are retained within the cell for they cannot pass through the pores According to the laws governing the distribution of electrolytes on both sides of a semi permeable membrane an equilibrium is established between the negative ions within the cells and the positive ions outside Positive and negative poles are close together the positive (source) on the outer surface of the cell the negative (sink) on the inner surface The close approximation of a positive and a negative pole is called a doublet (couplet or dipole)

If the cell rests no potential is measurable in the surrounding tissue because positive and negative poles balance each other an electric equilibrium exists Stimulation of the resting cells leads however to depolarization The permeability of the membrane changes and the negative ions emerge from the cell and cause a negative potential on its surface Owing to fundamental vital processes the membrane then again becomes polarized a process which is known as repolarization The resting normal condition is re-established

During depolarization (activation) and repolarization (recovery) of a muscle bundle potentials are formed which may be considered to originate at the boundary between depolarized and polarized tissue A part of the potentials thus created reaches the surface of the body

It has been found that with the spread of the excitation wave (depolarization) in the heart potentials are transmitted through the body so that an effect is created as if doublets spread with the positive pole (source) preceding the negative pole (sink) (Craib

Wilson and co workers, Ashman) The situation is much the same as if a battery wanders with its positive and negative poles close together The arrival of a negative potential is preceded by a positive potential If the depolarization wave spreads over the heart, those areas towards which the wave is advancing show a *positive potential while the areas behind the excitation wave show a* negative potential Both poles are close together at the boundary between polarized and depolarized tissue

During repolarization, potentials are again formed at the boundary between repolarized and depolarized tissue, but the poles are reversed The repolarization process proceeds slower and the the sum of all potentials created at any given moment is smaller

No electrical field is generated when the entire muscle is either completely activated or completely recovered

In important investigations the resting and the action potentials inside the axon cylinder of the giant nerve fibre of the squid were measured (Curtis and Cole Hodgkin and Huxley) These studies revealed that the action potential is about twice as large as the resting potential The negativity during activation is therefore not simply due to a change of membrane permeability Furthermore the action potential is much greater than the injury potential The difference in potential between the outside and inside of the fibre during rest (resting potential) is about 40 to 50 mV with the outside positive to the inside Activation is *accompanied by a reversal of this polarity so that the inside is more* positive and the difference in potential measures 95 to 168 millivolts These results have been confirmed on the single muscle fibre of the frog s ventricle (Woodbury Hecht and Christopherson) The average membrane resting potential was found to be 64 5 mV while the average action potential amounted to 77 2 mV

The finer mechanism leading to this difference in resting and action potentials is unknown

THE ELECTROGRAM AND THE ELECTROCARDIOGRAM

Classic and Doublet Hypothesis

If a strip of muscle is stimulated at one end (A) by means of a shock from an induction apparatus the muscle is excited or depolarized at this point (Fig 1a) The excitation is then rapidly transmitted over the entire strip toward the other end (B)

According to the classic theory of physiology every excited portion of the muscle becomes electronegative in relation to its unexcited part just as in a battery the zinc electrode becomes electronegative to the copper electrode If the two ends of a conducting wire are applied to the ends A and B of a muscle strip

(Fig 1a) after A is excited a current flows in the wire from B to A from the site of higher to lower potential If a galvanometer is included in the circuit it will register a deflection in a definite direction When this deflection is recorded graphically a wave is obtained which is directed upward (positive) providing the connections to the galvanometer have been made with the correct polarity (Fig 2)

If the excitation has affected the entire strip between A and B these points are equally excited then the same potentials exist

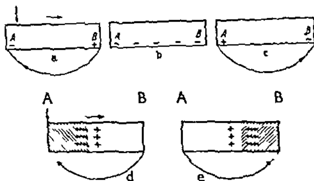


FIG 1 Diagram of spread of excitation in a strip of heart muscle Fig 1a b and c according to classic theory Fig 1d and 1e according to doublet theory

at both ends (Fig 1b) no current flows through the galvanometer the apparatus records no deviation and a zero (O) or iso-electric line is recorded

Recovery from the excitation begins very soon Naturally the excitation fades away earlier in the muscle end A which was stimulated first For this reason B now becomes more negative than A a current flows through the apparatus in the opposite direction (Fig 1c) and a downward directed (inverted or negative) wave is recorded (Fig 2) The doublet theory however which is well supported attributes the relative positivity of the end B to the fact that the excitation wave with its positive pole wanders towards it while A is negative because it faces the negative pole behind the excitation wave at the boundary between polarized and depolarized tissue (Fig 1d)



FIG 2 Electrogram from a strip of heart muscle

According to the doublet theory the poles are reversed during the repolarization A faces the positive pole while B faces the

negative pole at the boundary between depolarized and repolarized tissue (Fig 1e)

Accordingly, a diphasic tracing can be obtained from a muscle strip of suitable length and breadth with properly arranged leads, in it one wave is directed upward and the other downward, and the two waves are separated from each other by a short iso-electric line Such a tracing, obtained by *direct* leads from an excited muscle, is called an *electrogram* or Eg

Knowledge of this simple electrogram of a muscle strip facilitates the understanding of the more complicated currents of cardiac action obtained in man The first major difference consists in the fact that the human heart is composed of a very large number of muscle bundles varying in size, running in diverse directions, and interweaving in manifold ways A second and very important difference lies in the type of lead employed In clinical examinations the electrodes cannot be applied directly to the heart in order to record the tracing

During the spread of the excitation wave over the human heart potentials appear They face different directions and partly neutralize each other Only a fraction of the potentials reaches the surface of the body where they can be intercepted and measured This type of lead is *indirect*, and in contrast to the electrogram the tracing is called an *electrocardiogram* or Ecg

While the amplitude of the deflection in the electrogram depends somewhat upon the width and thickness of the muscle strip and is proportional to it this relationship is absent in the electrocardiogram In the latter only that fraction of the potentials formed by the heart which can reach the body surface is recorded

The electrical field created by the spread of the excitation wave is greatest in the area facing the direction of the spread The magnitude of the potential created by the excitation shows an inverse relationship to the square of the distance from the focus of origin of the potential Thus large potentials found on the cardiac surface diminish rapidly in size when measured farther away

In the electrocardiogram the position of the heart in relation to the sites of the electrodes and the status of the tissues surrounding the heart exert a very marked influence on the potentials which reach the surface of the body These influences have been studied in detail by numerous model experiments

Potentials developing along an axis which is perpendicular to lines joining the leads do not exert any influence on the electrocardiogram recorded in this lead

In these brief remarks the physical basis of the electrocardiogram has been intentionally oversimplified

THE ELECTROCARDIOGRAPH

Principle of Apparatus

If the thorax and pericardium of a dog are opened and the nerve of a nerve muscle preparation is applied to the surface of the exposed heart the muscle innervated by the nerve contracts with the same rate and rhythm as the heart (Kölliker and Muller 1856) In this simple and superb way it was proved that bio electric currents are produced by cardiac action

The following observation is another example of the same fact during an animal experiment contractions of the left half of the diaphragm are often noted which have exactly the same rhythm as the heart they vanish when the left phrenic nerve is lifted or cut below the point at which it touches the heart This also proves the development of electrical potentials by cardiac action

Waller studied and also measured those potentials which reach the surface of the body Owing to the inertia of his apparatus the capillary electrometer accurate records could not be obtained This goal was reached when Einthoven invented the string galvanometer (1903)

The principle of the apparatus is simple and generally known if a magnetic needle is suspended in the vicinity of a circuit through which a current is flowing the needle is deflected the amplitude and direction depending on the strength and direction of the current If a powerful fixed magnet is employed and a fine movable conductor is brought into its field the conductor will be deflected when a current flows through it The second principle is employed in the string galvanometer A thin metal thread or a quartz fibre coated with metal (the string) is suspended between the two poles of a powerful electromagnet The action current of the heart led from the person under examination is conducted through this string whereupon its movements correspond to the direction and strength of the current These movements are magnified many times by a microscope and are projected into a camera where they are photographed on a film or strip of paper A small slit with a lens in the camera aperture makes it possible to project the movement of a small section of the string on photosensitive paper

Formerly the apparatus was quite cumbersome and difficult to transport since the magnet required considerable space and had to

of each foot in the human being have atrophied The body odour at the pubertal age is due to the activity of the glands in the skin The sebaceous and apocrine glands become mature and produce secretion only after puberty in the human being This body odour becomes aggravated during menstruation in the female and in seborrhoeic subjects in both sexes

various conditions In the stratum corneum the bulla is formed in impetigo and in scabies But in scabies there are two bullæ, one in the stratum corneum and the other in the stratum mucosum like a dumb bell In the stratum mucosum again when a bulla is found at the upper part it may be due to herpes simplex or eczema, when in the middle of stratum mucosum it may be due to herpes zoster or varicella while in the lower part of the stratum mucosum the bulla may be due to epidermolysis bullosum or pemphigus Sub epidermal bulla is found in dermatitis herpetiformis where the bulla develops under the stratum basalis and above the dermis

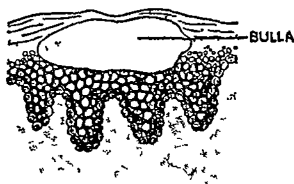
Cyto diagnosis Is done by preparing a smear on a glass slide from the floor scraping of an unruptured bulla and staining with leishman stain or Giemsa's stain The cytology of the bulla floor is helpful in the diagnosis of various skin diseases Large number of eosinophil cells with clumps of normal epidermal cells are found in the floor of dermatitis herpetiformis Enlarged giant epithelial cells called baloon cell are found in the scraping examination from the bulla floor of herpes simplex, herpes zoster and varicella Absence of cellular elements is seen in the cytological smear examination of epidermolysis bullosum Lysis of the epidermal cells with pyknotic nuclei are found in the cytological smear examination of a pemphigus bulla (Tzanck test) In erythema multiforme floor smear examination shows large number of polymorphonuclear leucocytes with normal cells in the epidermis Tzanck test is helpful to differentiate pemphigus by the presence of its acantholytic cells from dermatitis herpetiformis and epithelioma

(g) **Wheal**—Is a transitory elevated red lesion with a whitish centre Seen in urticaria

(h) **Vesicle**—Is a circumscribed pin-head sized swelling in the skin containing fluid Seen in eczema and pompholyx

(i) **Bulla** - Is a circumscribed swelling in the epidermis containing fluid and is larger in size than a vesicle Examples are herpes simplex herpes zoster, impetigo, pemphigus

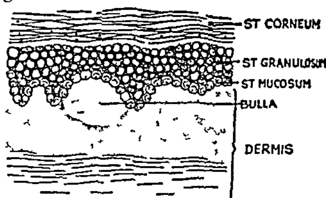
The situation of the bulla in the skin is helpful in the microscopical diagnosis of a skin disease Bulla may be (a) intra epidermal (Fig No 7) and (b) subepider



BULLA HIGHER UP
IN EPIDERMIS

Fig No 7

mal (Fig No 8) Intra epidermal bulla is found in



SUB EPIDERMAL BULLA
Fig No 8

The Secondary skin lesions are formed as a result of the degeneration of the primary skin lesions due to bacterial invasion and other causes —

(a) **Scales**—Are the dry exfoliation of the epidermal layer of the skin It is seen in ringworm, pityriasis versicolor and psoriasis Due to intra epidermal edema there is interference with the cell nutrition of the epidermis The stratum granulosum is incompletly developed and the cells are incompletly keratinized and the nuclei persist in the cells The process is called *parakeratosis* Is seen in eczema and psoriasis When there is localized or diffused hypertrophy of the stratum corneum it is called *hyperkeratosis*

(b) **Scab**—Is a dried mass of serum with scales, blood cells and organisms It is seen in impetigo

(c) **Excoriation**—Is the condion of the skin caused by the removal of epidermis from the skin Is seen in abrasions

(d) **Fissure**—Is a linear excoriation deep down to the dermis and is painful Seen at the angles of the mouth and in the groins

(e) **Ulcer**—Is a circumscribed lesion characterised by the loss of epidermis and a portion of dermis It is seen in varicose ulcer

(f) **Scar**—Is the new formation of fibrous tissue in the dermis There is no scar formation when the epidermis only is involved Scar tissue has no hair follicle

(g) **Pigmentation**—Occurs in the skin as a result of inflammation due either to increase in the melanin or due to extravasated blood

(j) **Pustule**—Is a bulla filled with pus A pustule is formed generally in association with a hair The pustule may develop at the opening of a pilo sebaceous follicle as in Bockhart's impetigo and at the bottom of a hair follicle as in furunculosis When many such hair follicles are affected there occurs a phlegmonous degeneration forming multiple pus points like a honey comb Such a condition is called a carbuncle

(k) **Abscess**—Is a large pustule There are two different types of abscesses such as (i) ordinary abscess which is but a large sized pustule and the other is (ii) the micro abscess which can be seen in the stratum corneum in psoriasis and is called micro abscess of Munro, and also the intra epidermal abscess of pemphigus vegetans

(l) **Cyst**—Is a non inflammatory pustule When filled with sebaceous material it is called a sebaceous cyst

✓ (m) **Comedo**—Is a black grain shaped plug at the mouth of a hair follicle which is made of sebum and cells of stratum corneum Comedo is seen on acne vulgaris

✓ (n) **Burrow**—Is a tunnel formed by the female sarcoptes scabiei in the layers of epidermis above the rete mucosum The dead acarus is always found at the bottom of this tunnel This is typical of a scabies lesion

Collagen fibres or the elastic fibres may degenerate

(ii) **Allergy**—Is the specific hypersensitiveness of an individual to foods, drugs bacteria and physical agents Allergy is a biological reaction of the body due to some skin disease and this allergy is an alterd capacity of the tissues to react to a substance Skin is an indicator of systemic allergic diseases

Various skin diseases are due to allergy Specific hypersensitiveness is always hereditary **Atopic dermatitis** is an example of such a specific hypersensitiveness in an individual The sensitizing substances are termed **atopens**

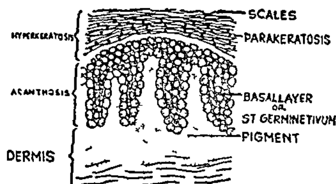
Body may be sensitized to any substance with which it comes in contact like chemicals, proteins plants or flowers and sometimes to infection such as pyogenic infections and to fungus or tubercular infections When the body is sensitized by contact or by absorption there develops on the skin a rash which is called the 'id' reaction or secondary rash

In case of bacteria it is called **bacterid,** in case of fungus it is **trichophytid,** in case of tuberculosis it is called **tuberculid** This 'id reaction is a specific allergic sensitization to some toxin which is absorbed in the circulation

Types of allergy (1) **Food allergy**—some individuals are sensitive to certain food stuffs This specific hypersensitiveness to food stuffs is inherited in that particular individual Types of foods that cause sensitization are protein foods non protein foods, organic foods and inorganic foods Food sensitization is commonly seen during the second year of life This type of sensitization to foods is a factor in the production of atopic eczema

(h) **Hyperkeratosis**—Is the thickening of the stratum corneum e g corns (Fig No 9)

(i) **Parakeratosis**—Is the presence of incompletely cornified, edematous and nucleated cells in the stratum corneum e g Eczema, psoriasis (Fig No 9)



THICKENING OF SKIN LAYERS
Fig No 9
Section of Skin (Diagramatic)

(j) **Acanthosis**—Is the thickening of the stratum mucosum e g warts, psoriasis (Fig No 9)

(k) **Lichenification**—Is characterised by papular, smooth and variously shaped skin lesions aggregated together with exaggeration of the normal skin markings showing a criss cross pattern of the lesion Develops as a result of long continued itching e g Lichen simplex chronicus (Widal)

(l) **Atrophy**—Is characterised by the thinning of the epidermis May result from wasting diseases, due to old age, lupus erythematosus, morphea etc

(m) **Dermic degeneration** Is *of four types* e g fatty, hyaline, mucoid and edematous

(8) Blood sugar estimation (fasting)

(9) Blood cholesterol estimation (fasting)

(10) Blood calcium estimation

(11) Blood Vitamin A or C estimation (fasting)

(12) Blood protein estimations

(13) Mantoux test

(14) Sensitivity test

(15) Skiagram of chest and bones

(16) Biopsy and histopathological examination Biopsy material is preserved in a 10 per cent formaline in normal saline solution

(17) Skin scraping is examined for fungus with a 10 p c aq sodium hydroxide sol on a slide with a cover slip which is warmed on a Bunsen burner and the nail clippings are boiled with 40 per cent sodium hydroxide in a test tube for fungus examination

(18) Culture of skin scraping and nail clippings for fungus or bacteria in different media

(19) Cytology—fluid or the scraping of the floor of an unruptured bulla is examined for different types of cells In Tzanck test the smear on slides is stained with Giemsa s stain when acantholytic cells are seen in case of pemphigus

(20) Serology—blood W R and Kahn tests

(21) Animal parasites such as the louse or its eggs and the sarcoptes scabei are removed and are put on a glass slide with a drop of saline and are examined under microscope

Hence crab, prawn, egg and such other foods are discarded in patients suffering from atopic eczema

(2) **Drug allergy**—Some individuals are sensitive to some drugs Drug allergy is commonly seen in arsenic, sulpha drug, antibiotic and such others Hereditary factor for this specific hypersensitiveness is present The mechanism of drug allergy is anaphylactic in nature Drug allergy is specific and s permanent Quite a large number of skin diseases are due to drug allergy Dermatitis venenata, contact dermatitis, dermatitis medicamentosa and fixed drug erruptions are examples

(3) **Bacterial allergy**—fungus infection, pyogenic bacterial infection, syphilitic infection and tuberculous infection in a person may develop from sensitization in an individual

(4) **Physical allergy**—When symptoms are caused by cold, heat and light they are said to be due to physical allergy

INVESTIGATION OF A CASE OF SKIN DISEASE

(1) *Examination of stool after a saline purgative*
(2) Examination of urine
(3) Blood picture—Total R B C and W B C, differential count and hæmoglobin p c
(4) Erythrocytic sedimentation rate
(5) Examination of sputum
(6) Skin snip smear examination for *Bacillus* Hansen, Leishman donovan bodies etc
(7) Nasal smear for Bacillus Hansen

CHAPTER III

CARD OF A CASE

Date of first visit — Disease
Name — Address
Age — Sex
Referred by Dr
Complaints with duration
History

Family History
Personal History—Past
Present

General Examination—Hairs on head rash on fore head eyelids face and neck Jaundice or anaemia Tongue teeth cervical axillary epitrochlear and inguinal glands and nails

Respiratory system
Cardiovascular system

Skin lesion

Investigation

(1) Examination of Urine
(2) Examination of Stool
(3) Examination of Blood for total differential counts Hb p c and parasites
(4) Erythrocytic sedimentation rate
(5) Skin scraping examination under microscope for fungus or A F B
(6) Skin scraping for culture
(7) Blood chemistry
(8) Blood proteins
(9) Blood vitamins
(10) Biopsy
(11) Mantoux test
(12) Skiagram of chest and bones

Treatment
Follow up

(22) Diascopy—when a glass slide is pressed on a skin lesion the part is exsanguinated Apple jelly appearance is seen in case of lupus vulgaris on diascopy

(23) Wood's light—the fluoresence is caused on the nail, skin or hair when exposed to the ultra violet light which is filtered through a glass plate containing nickel oxide This Wood's light is of special value in the diagnosis of different types of ringworm infections Infected hairs with Microsporon fluoresce green whereas hairs infected with Trichophyton do not fluoresce Wood's light is also used to examine the urine of porphyrin which gives a pinkish to red fluorescence

Chapter III

CARD OF A CASE

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(11) Mantoux test
(12) Skiagram of chest and bones

Treatment

Follow up

Chapter IV

TREATMENT OF SKIN DISEASES

In the tropical country it is always essential to treat the patient for any parasite in the gastro intestinal system e g amæbiasis, giardiasis, oxyuriasis, ascariasis bacillary dysentry, ankylostomiasis etc, blood infections like malaria etc, other infections like visceral kala azar, tuberculosis, nutritional deficiencies and blood diseases

Internally

(1) (a) Arsenic—Liquor Arsenicalis (Donovon's solution) dose m 5, may be used in increasing doses, (b) Pentavalent arsenic for injection such as Acetylarson, N A B, Thiosermine (Brahmachari)

(2) (a) Penicillin crystalline 'G'' aquous solution is injected intramuscularly in dose of 200,000 I U (0 2 mega unit) twice daily for 5 to 7 days, (b) Penicillin orally is some times used but does not have much effect in acute conditions, (c) Penicillin locally should never be used as it produces sensitization

(3) Streptomycin gram 1 by intramuscular injection Where patient cannot tolerate it Dihydrostreptomycin gram 1 is injected together with PAS by mouth

(4) Aureomycin (250 mg) capsule is given orally 6 hourly for 4 days usually with Vitamin B Complex by mouth 3 to 4 times after food

(5) Terramycin (250 mg) is given orally every 6 hours for 4 days with vitamin B Complex

(6) There are other antibiotics also e g Ilotycin Acromycin etc

(7) BAL (British Anti Lewisite) known as Dimercaptol 2 5 mg per Kilogram of body weight is used by intravenous injection This may also be used as a 3 p c ointment

(8) Antimony as stibanate (Gluconate) intramuscularly or Ureastibamin (Brahmachari) intravenously or Stibinol (Brahmachari) or Stibatin (Glaxo) are used by intramuscular injections

(9) Sulphonamide such as Sulphadiazine, Elkosin etc, 0 5 gram is given orally every 4 to 6 hours for 4 to 7 days May also be given intramuscularly in acute conditions

(10) Sulphone such as D D S (B C P W), D A D P S (I C I) Novotrone (Bengal Chemical) Sulphetrone (B W), Thiosemicarbazone (A D) 50 mg daily, Diasone (Abbott) 0 3 gm daily as tablets are given orally in graduated increasing doses Injections of sulphetrone or novotrone solution are given intramuscularly

(11) Vitamins may be used together or separately by oral method and also by intramuscular route in high dose

(12) Liver extract is used both orally and intramuscularly

(13) Iron as Ferrous Sulphate is given by mouth

(14) Emetine hydrochlor is injected in one grain dose intramuscularly for 6 consecutive days with rest

(15) Enterovioform (Ciba), Enteroquinol (East India Pharm Works), Siosterin (Geigy), Aralis (Winthrop) are given by mouth

(16) Methionine as Neomethidine (Neo Pharma) is used orally as tablet or *syrup* and as solution intramuscularly or intravenously by injection to improve liver function

(17) Cortisone and ACTH are used for fatal skin diseases, incapacitating skin diseases, drug reactions, erythema multiforme and rarely in psoriasis with arthropathies, dermatomyositis and urticaria Should be used very cautiously This therapy should not be stopped suddenly as it produces withdrawal symptoms charac terized by exacerbation of the original symptom Cortisone suppresses the internal *production of the adrenocortical hormone* causing adrenocortical deficiency

(18) Isoniazide (Isonicotinic acid hydrazide) is used in cutaneous tuberculosis It is relatively non toxic May be used in combination with other anti tuberculous drugs

(19) Tar is commonly used in treating skin diseases As an antipruritic in 2 per cent solution as Liq Picis carb detergens and may be used with Lotio calamine or with an ointment Crude coal tar as 10 p c crude coal tar in Acetone is used for painting lichenified areas Prag mater (S K F), Primo derma (Primco) may be locally used as antipruritic and also for treating lichenified lesions of the skin

(20) Refrigeration is done by solid carbon dioxide It is used from 1/2 to 2 minutes Commonly used for infective warts, moles superficial rodent ulcers and the like

(21) Ultra violet light is used in the treatment of various skin diseases

(22) X ray therapy, both superficial and deep, is used in malignant and other skin diseases

(23) Radium, as needles or plaques, is used in malignant skin deseases and haemangiomas

(24) Electro cautery is used for the removal of warts, cutaneous tugs, and small malignant growths Electro cautry needle is used for electrolysis and is commonly used for epilation of hairs in hypertrichosis

(25) Hyalurannidase is sometimes used in the treatment of keloid

(15) Enterovioform (Ciba), Enteroquinol (East India Pharm Works), Siosteran (Geigy), Avlalis (Winthrop) are given by mouth

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(iii) Ichthyosis hystrix is shown by localized exaggerated hypertrophy with dryness of the skin. The hypertrophic skin generally forms bands on the extremeties.

Signs and symptoms. Dry and rough skin is known as xeroderma. Patients feel dry. There is no sweating particularly in winter. When this condition is exaggerated with the appearance of fish scale like (Fig No 10) condition of the skin of the whole body except the flexures sometimes with a streak of pigmentation at the fringe of the scales is called ichthyosis. This condition gets worse during winter. Some of the patients suffering from ichthyosis develop localized band of hyperkeratosis on the upper and lower extremities called ichthyosis hystrix. Patients become usually dark.



Fig No 10
Ichthyosis (Boy of 6 years)

Patients seek cold and dry corners in a room. Some of these patients also suffer from night blindness. Itching is sometimes a troublesome symptom. Itching causes scratches on the skin through which pyogenic organisms gain entrance and infective eczema sets in.

Diagnosis. (1) History of dry skin since childhood, (2) Clinical examination reveals dry, pigmented skin with

CHAPTER V

CONGENITAL DISEASES OF THE SKIN

1 Ectodermal defect
2 Ichthyosis
3 Epidermolysis Bullosum
4 Urticaria Pigmentosa
5 Ehler-Danlos Syndrome

ECTODERMAL DEFECT

These are as follows —

(1) Pachyonychia congenita is the absence or defect of nails
(2) Absence of sweat glands
(3) Dental dysplasia
(4) Presence of nevus.
(5) Cutis vertices gyrata
(6) Supernumary finger and supernumary nipple

ICHTHYOSIS

Definition Ichthyosis is a congenital skin disease characterized by dry fish scale appearance of the skin

Etiology Is quite common in the tropics Supposed to be hereditary as it is sometimes found in more than one member of a family Recently it has been found to be *associated with Vitamin A deficiency* Acquired ichthyosis is found in patients suffering form Hodgkin's disease and nutritional dificiencies The disease commences at the prepubertal age Both sexes are equally affected

Varieties (i) Xeroderma is a mild hypertrophic condition with dryness of the skin, (ii) Ichthyosis is characterised by hypertrophy of the skin with dryness, fish scale appearance and pigmentation of the skin,

Treatment Prophylaxis patient should stay in places of equable climate and must avoid severe winter and should take vitamin A rich food

General treatment consists of applying locally oil all over the body before bath as is the practice in the tropics Olive oil is helpful Some patients are happy with the local application of a mixture containing equal parts of glycerine and water

Vitamin A is given in high doses of 50,000 international units twice daily by mouth throughout the winter and thereafter once daily with short intervals

Diet should consist of carrots butter, ghee, milk liver, meat etc

EPIDERMOLYSIS BULLOSUM

Definition Is a congenital skin disease characterized by the development of bullae over the joints of the extremities by the slighest injury

Etiology Is found in the tropics Found in several members in a family

This condition is supposed to be associated with faulty porphyrin metabolism Occurs in both the sexes Sometimes Vitamin C deficiency is associated also

Varieties (i) Simple type (ii) Dystrophic type

Signs and symptoms The **simple type** occurs after birth Bulla is found with very mild injury on any part of the body The condition gets well at puberty

fish scale appearance, (Fig No 11) (3) Defective dark adaptation, (4) Low blood vitamin A (5) Histopathology



Fig No 11
Ichthyosis (Boy of 12 years)

shows thinning of the epidermis with loss of wavy outline at the dermo epidermal junction Atrophy of sweat glands Thickening of the stratum corneum with absence or marked thinning of the stratum granulosum

Differential diagnosis (1) Tuberculoid leprosy, (2) Seborrhoic dermatitis, (3) Besnier's prurigo

Prognosis Is not good so far as the cure is concerned Death is not the common termination of the disease Vitamin A therapy improves the conditio

Diagnosis (1) History of the development of bullae even with very mild injury at the places of



Fig No 14
Epidermolysis Bullosum

trauma, (2) Clinical examination reveals bullae formation pigmentation and hypertrichosis (3) Porphyrin estimation in urine shows increased porphyrin excretion (4) Histopathology shows intraepidermal or subepidermal bulla formation There is hyperpigmentation in the stratum basalis Dermis shows rupture of the elastic tissue

Differential diagnosis (1) Pemphigus, (2) Drug rash (bullous type)

Prognosis Simple type gets well at puberty but the dystrophic type is never cured

Treatment Prophylaxis consists in careful handling of the patients to avoid injury or pressure over the traumatic areas and vitamin C (200 mg) 2 to 3 times daily by mouth can be given

Treat the ulcer when develops with 2 p c Hydrarg Ammon ointment locally

The **dystrophic type** shows bullae developing after injury or irritation on the legs (Fig No 12) and hands, toes, ankles, knee joints and fingers Wrist and elbows are also involved Repeated bulla formation with eczematization occurs at the finger ends (Fig No 13)



Fig No 12
Epidermolysis Bullosum



Fig 13 Epidermolysis Bullosum

The bulla fluid looks serous or mixed with blood *Bullae* get ruptured and leave scars, pigmentation and and cysts in groups The bulla may appear inside the mouth also Nail changes may be present Nail becomes birttle, rough and discoloured Hyper trichosis is sometimes an associated feature

pigmented itchy macules all over the body (Fig No 17) (3) Histopathology shows infiltration with mast cells (mast cells are cuboidal in outline) in the dermis Excessive melanin pigment is found along the stratum basalis with thinning of the epidermis In the bullous type the bulla is situated intraepidermally and a dense mast cell infiltration is found in the dermis below the stratum basalis



Fig No 16
Urticaria Pigmentosa
(Back view of a girl aged 7months)

Differential diagnosis (1) Urticaria (2) Fixed drug rash (3) Xanthoma, (4) Incontinentia pigmenti

Prognosis Patients suffer throughout childhood Sometimes subsides at puberty or in adult life but the pigmentation is permanent



Fig No 17
Urticaria Pigmentosa
(Boy aged 6 years)

Treatment Locally antipruritic lotions are used such as Liniment Calamine with 1 per cent Phenol or an ointment containing —

Locally hydrocortone gives temporary encouraging results and cortisone orally is helpful when the patient is in distress

URTICARIA PIGMENTOSA

Definition Urticaria pigmentosa is a congenital skin disease characterized by chronic, itchy and pigmented papules on the skin

Etiology Is not unknown in the tropics Cause is not known Some believe that it is due to the disturbance of some endocrine glands There are others who believe it to be a congenital blood dyscrasia Both sexes are affected but is common in males Age of onset is usually before the sixth month of life

Signs and symptoms Starts in the first year of life (Fig No 15) Starts as macular or papular urticaria which is itchy but later on redness subsides and yellowish or blackish pigmented macules persist The lesions may occasionally be bullous or nodular



Fig No 15
Urticaria Pigmentosa
(Front view of a girl aged 7months)

Five types of lesions are seen — (1) Macular, (2) Papular, (3) Maculo nodular, (4) Nodular, (5) Bullous At puberty it gets apparently cured but reappears after puberty Distribution is all over the body (Fig No 16)

Diagnosis (1) History of urticarial wheals starting early in infancy, (2) Brownish or blackish

Diagnosis (1) History of hyperstretchability of skin and exaggerated movements of joints since birth, (2) Skiagram of long bony ends shows abnoramal epiphysial structure (3) Histopathathology shows excessive formation of elastic fibres in the dermis



Fig No 18
Cutis Hyperelastica
(Case of Major N N Chakravorty A N Banerjee and S Ghosh)

Differential Diagnosis Cushing s syndrome
Prognosis Is not a curable disease
Treatment No treatment is of any use

Acid salicylic	gr 10
Menthol	gr 5
Vaseline alba	oz 1

X'ray therapy is sometimes helpful

Internally—Antihistaminics may be tried Autohaemotherapy starting with 5 c c blood intramuscularly twice weekly and increasing by 0 5 c c to 10 c c, 12 such are helpful Vitamin C (500 mg) intramuscularly is injected twice daily for a week or so also may help Adrenocorticotropin (ACTH) therapy in 20 units aquous *solution by intramuscular injection* every 6 hours or ACTH gel 20 units twice daily gives temporary good result DOCA (Desoxycorticostrone) 4 mg as sublingual tablet daily for 6 months has been advocated

EHLER DANLOS SYNDROME

(OR CUTIS HYPERELASTICA)

Definition Ehler Danlos syndrome is a congenital skin disease characterized by hyperextensibility of the normal skin

Etiology Cause is not known Sometimes more than one member in the family suffer Both sexes are affected Starts from birth

Signs and symptoms The skin of any (Fig No 18) part of the body can be stretched to a great length Joint movements are exaggerated (Fig No 19) and wrist and finger joints are hyperextensible There may be found ecchymosis on the extremities Hyperextensibility of the abdominal skin may be found Fine perchment like appearence of the skin is seen on hands and feet Pads develop on elbows and knees (Fig No 20) In women clitoris may be enlarged

CHAPTER VI

NON INFECTIOUS INFLAMMATORY SKIN DISEASES

Non infectious inflammatory skin diseases form a group with an allergic background and is characterized by inflammation and itching

Classification 1 Eczema
2 Dermatitis

ECZEMA

Definition Eczema is a non infectious inflammatory syndrome characterized by itching and inflammation of the epidermis

Etiology (1) Irritant may be a cause such as chemicals when it is called **contact dermatitis** or **industrial dermatitis**, (2) Allergen may be a cause Allergen may be of vegetable origin or animal origin when it is called **allergic dermatitis**, (3) Internal toxin may cause such as from food or auto infection when it is called **internal toxic dermatitis** (4) Physical causes may lead to eczema such as burns from any source and is called **dermatoses due to physical causes**

Classification of eczema (Modified from Percival s classification)

1 Eczema due to burns
2 Eczema due to contact
3 Eczema due to infection

such as follicular, flexural and post traumatic

4 Eczema due to varicose vein
5 Atopic eczema

such as infantile eczema, Besnier s prurigo, nummular eczema and lichen simplex chronicus (Widal)



Fig No 19
Cutis Hyperelastica
(case of Major A N Chakraborty
A N Banerjee & S Ghosh)



Fig No 20
Cutis Hyperelastica
(case of Major
A N Chakraborty
A N Banerjee and S Ghosh)

different chemicals in the laboratory of the medical chemistry, and in handling different medicines (Fig No 21)



Fig No 21
Dermatitis
(Contact dermatitis due to hypo in a photographer)

in the practical pharmacy laboratory Common amongst the practising medical practitioners who have to handle different antibiotic solutions for injection Amongst the Indian ladies who put vermillion (Fig No 22) on their forehead as a cosmetic and those who use Sandel wood paste on forehead as a religious cosmetic (Fig No 23) or alta these types of chemical eczemas are of common occurrance (Fig No 24) Amongst ladies it is commonly found after the use of different cosmetics



Fig No 22
Contact Dermatitis Forehead
(Due to vermillion used as a cosmetic)

ECZEMA DUE TO BURN It is not rare Sun light causes this type of eczema The fireman in the engine, the cool, those who handle ultra violet rays, X rays, radium, tracer substances are likely to get eczema due to burns Even when the skin is burnt by fire there may develop eczema The sunlight causes eczema alone when the skin is sensitized such as are

(a) Solar eczema,
(b) Hydroa aestivals,
(c) Xeroderma Pigmentosa,
(d) Drug sensitivity
(e) Sun's ray is responsible for the production of certain diseases such as are
(i) Pellagra, (ii) Lupus erythematosus, (iii) Sudamina, (iv) Keratosis

The lesions are erythematous which soon become papulo vesicular in type Sometimes the lesions may be bullous as in Hydroa aestivals

The skin lesion is very itchy and leave permanent scarring when incessantly scratched The most important predisposition to solar eczema is caused by the taking of sulpha drugs Pigmentary changes may also occur

Treatment Consists in avoidance of the direct exposure to sunlight and protection against ultra violet, x-rays, radium and the like Workers who have to expose themselves to fire should be properly protected In a developed case Lotio Calamine when frequently applied helps 10 p c Para Amino Benzoic Acid locally may help

ECZEMA DUE TO CONTACT It is very common all over the world and increases in a country with its industrialization It is seen amongst the non clinical medical students who have to handle the formalised bodies while doing dissection, while handling

as lipstick, rouge pomade, snow, nail varnish, plastic as spectacle frame (Fig No 25) or as hand bags and



Fig No 25
Contact Dermatitis Face
(Due to use of plastic spectacle frame)

ornaments and is known as **cosmetic dermatitis** Use of chromium plated articles (Fig No 26) and handing



Fig No 26
Contact Dermatit s
(Watch band dermatitis due the use of chromium pl_ted watch band)

chromium may cause contact dermatitis The workers in the industry have to handle different types of chemicals



Fig No 23
Contact Dermatitis Forehead
(Red sandal paste used as a cosmetic)



Fig No 24
Contact Dermatities Sole
(Due to cosmetic use of Alta)

(Fig No 29 & 30) which is becoming a problem in the developed countries of the world

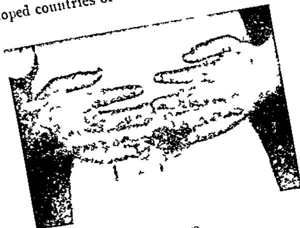


Fig No 29
Industrial Dermatitis
(Due to handling of varni h)



Fig No 30
Industrial Dermatitis Palms
(Due to handling of jute)

and oils which cause contact eczema and is called trade eczema or (Fig No 27 & 28) **Industrial Dermatitis**



Fig No 27
Industrial Dermatitis
(Due to plastic)

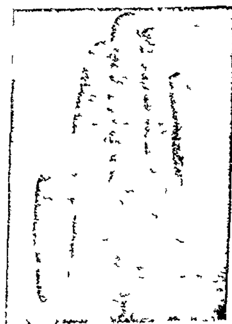


Fig No 28
Industrial Dermatitis
(Due to hypo in a photograph)

(Fig No 29 & 30) which is becoming a problem in the developed countries of the world

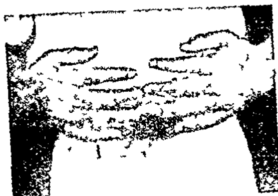


Fig o 9
Industrial Dermatitis
(Due to handling of varni 1)



Fig No 30
Industrial Dermatitis Palms
(Due to handling of jute)

Any type of acid, any alkali and all the different salts either alone or in combination are liable to produce contact eczema As an example carbolic soap may produce contact eczema Wrist watch bands made of plastic material or chromium plating produce contact eczema

Signs and symptoms In the early stage edema, erythema and vesiculations with no margin can be seen Generally seen on the uncovered parts of the body but may get generalised and can affect the scalp, eyelids and face In the chronic stage it resembles chronic eczema characterized by lichenification with itching or erythemato vesiculation Sometimes the parts become hyperpigmented and not infrequently leucoderma has resulted from this

Diagnosis (1) Careful history of work, hobby and the like, (2) Patch test is done using the suspected material In a positive case an eczema reaction is obtained in 24 to 72 hours, (3) Biopsy and *histo pathology* shows the formation of vesicle in the stratum mucosum with intra cellular edema of the epidermis and dilatation of the papillary blood vessels Later on there is acanthosis with parakeratosis and hyperkeratosis

Differential Diagnosis In the acute stage contact eczema has to be differentiated from *Erysipelas*, *Cellulitis*, Erythema multiforme and Dermatitis herpetiformis

Complication Is the 'id' reaction characterised by the development of an erythematous rash all over the body which on the palms and soles become vesicular The distribution is symmetrical and is itchy

Prognosis Is good when diagnosed and is treated properly

Treatment As a prophylaxis the offending material must be removed In the acute stage frequent application of (a) Lotio Calamine is very helpful Sometimes different types of lotions give good result such as

(b) Menthol	dr 4
Zinc oxide	dr 2
Glycerine pure	oz 1
Liq Plumbi subacetatis dil	oz 1
Liq Calcis	oz 20

Lotio for external use To apply every hour for a day or two

In the sub acute stage when there is much *oozing* soaks with 1 p c lotio silver nitrate frequently for 24 to 72 hours is helpful but when the oozing is non dripping 1 p c aquous gentian violet is painted When dry it is kept bandaged with 1 p c ung Ichthyol for about a week changing the dressings every day and cleaning with bland soap (Margo soap of Calcutta Chemical) and boiled warm water When chronic 2 p c Acid salicylic in a bland ointment (c) or 2 p c liq picis *carb* detergens or both may be used Bland ointment consists of

(c) Zinc oxide	dr 1
Pulv amylum	dr 1
Vaseline album	oz 1

Ung for external use

When dry and is left with a lichenified area 10 p c crude coal tar in acetone is painted once daily and is *dusted* over with a powder (Pearl powder—B C P W) and is kept bandaged for 24 hours with the bland ointment It is cleaned next morning with

warm water and a bland soap After drying the part with spirit rectificatus coal tar is again painted and dressings applied This treatment is not repeated for more than 6 times as tar is carcinogenic Even after this treatment a remnant *is left which is treated* with suberythema doses of x rays at fortnight's intervals with a low k v Worker must be kept off work

NAPKIN RASH

Definition It is a skin disease of the infants characterized by erythematous or erythemato squamous rash over the crural, genital, *perineal and gluteal regions*

Etiology It is a type of contact dermatitis Alkali is supposed to be the cause The urea in the stool and urine of the infant ferments into ammonia and is said to cause the napkin rash Sometimes it is thought to have a constitutional basis Common in infants for whom napkin is commonly used In the tropics napkin dermatitis is much less common as the napkin is changed soon after it gets soiled

Signs and symptoms It starts as an erythema but soon the lesion becomes papular and vesicular After sometimes the vesicles rupture giving rise to multiple ulcers Site it is confined only to the napkin area e g, in the crural, genital, perineal, gluteal and sometimes the medial sides of both thighs The erythemato papulo vesicular rash may spread down the legs to the heels or upwards over the back or abdomen

Diagnosis (1) Erythemato papulo squamous rash, (2) Site napkin area, (3) Age of patient infant

Differential diagnosis (1) Contact dermatitis to soap, (2) Congenital syphilitic cutis, *(3) Fungal infection*

Prognosis- Is good

Treatment

As a prophylaxis a powder may be dusted over the napkin area containing Zinc oxide dr 4
Pulv Bismuth subgallate dr 2
Pulv Calomel gr 15
Ft Pulv

Prophylaxis is to examine the napkins frequently and change it as soon as it is soiled After washing the napkin with soap and water it should be washed under running tap water and finally rinsed in a 10 p c aquous lotion of Boric acid and dried Sometimes lotio for external use is helpful Acid Tannic gr 15 in aqua destilata oz 1 Ft lotio for external use or "Tanofax" ointment (B W) may be used

Curative consists of washing the buttocks and napkin areas with boiled warm water and drying the parts before putting on napkin again Dilute solution of tyrothricin has been advocated for local use on the skin lesions Orally a powder may be given twice daily for 4 days such as Grey Powder gr ½ Ft Pulv for a dose

ECZEMA DUE TO INFECTION

This type of eczema is caused either by strepto staphylo yeast or fungus These are further classified under 3 groups such as—

(a) Flexural infective eczema,
(b) Follicular infective eczema,
(c) Post traumatic infective eczema,

Flexural infective eczema may be erythemato vesicular or vesiculo squamous lesions distributed on the flexural surface. (Fig No 31) such as behind the ears, on the eye lids, below the chin in the axillæ, below the

pendulous breasts, in the umbilicus, in the groins, in the natal cleft and in between the fingers and toes



Fig No 31
Flexural Infective Eczema

In follicular infective eczema the hairy regions are involved such as the beard region when it is called "sycosis barbae" (Fig No 32), the nuchal region when it is called "sycosis nuchae", pubic region and the limbs (Fig No 33) The Scalp of infants may be affected causing eczema of infants (Fig No 34) which is different form infantile eczema.



Fig No 32
Sycosis Barbae

Rarely the follicular infective eczema produces a



Fig No 33
Follicular Infective Eczema
(Dorsum of feet)



Fig No 34
Eczema of Infant
(Face is free)

band like lesion on a limb which may be linear or spiral in shape This type of eczema is known as **Lichen striatus**

Post-traumatic infective eczema is commonly seen amongst masons, miners, and amongst those whose works involve handling of dusts Erythematous or erythemato squamous lesions are found on the limbs extending from the ankles (Fig No 35) and



Fig No 35
Post Traumatic Infective Eczema
(Showing chronic edema of one leg)

wrists to the knees and elbows Post traumatic infective eczema is also found on the nipples Eczema of nipples is common amongst mothers who are nursing their babies (Fig No 36)



Fig No 36

Eczema of Nipples and Breasts

(Case of Captain S N Roy)

Treatment Removal from the place of work is a prophylactic measure if it is a dusty occupation Treatment is given according to the stage of the disease If it is an acute condition lotio calamine is applied at frequent intervals In the sub acute stage when there is dripping oozing 1 p c aquous silver nitrate solution soaks are applied In the non dripping subacute stage 1 p c aqueous gentian violet is painted followed by 1 p c Ichthyol ointment and is kept bandaged In the chronic stage is used 2 p c ung Acid salicylic or 2 p c Liq picis carb det or both in a bland ointment containing Zinc oxide and Pulv Amylum each dr 1 in an ounce of vaseline alba This ointment is applied for a week Then if need be 10 p c crude coal tar is painted once daily for

4 to 5 days At the end x ray therapy for 3 to 4 times at fortnightly intervals will cure the condition

ECZEMA DUE TO VARICOSE VEIN

Etiology The cause of the eczema is not known Inherent weakness of the venous system may be responsible for the eczema

Predisposing causes in women are chronic constipation, pelvic tumour, pregnancy and the like Common amongst those who keep on standing long periods Common in both sexes

Signs and symptoms Tortuous prominent veins are seen on the calfs, on the knee joints and along the thighs Oozing round about the ankles may be the earliest sign followed by erythemato squamous lesions which on cleansing shows patchy denudation of the epidermis A punched out ulcer may be produced round about the ankle surronded with erythemato squamous and eczematous lesion with pigmentation (Fig No 37)



Fig No 37
Varicose Eczema

Prognosis is good

Treatment is by rest and elastoplast dressings to the varicose veins Injection of sclerosing fluid in the veins is also done

Eczema is treated with 1 p c aquous Silver nitrate solution soaks for 2 days and then by 1 p c aquous gentian violet followed by 1 p c ung Ichthyol and bandaged over with elastoplast dressing

ATOPIC ECZEMA This is a constitutional skin disease The factors which are responsible for the causation of atopic eczema are heredity, over anxiety and a sense of insecurity Onset or relapse of the disease occurs with the psychological upsetting Sometimes there is a hypersensitivity to some known allergens The allergy may be present to particular foods, to different bacteria or to intestinal parasitic infections which are so common in the tropics

Predisposing factors are anaemia, malnutrition and septic focii

Classification (1) Infantile eczema
(2) Besnier s prurigo
(3) Nummular eczema
(4) Lichen simplex chronicus (Widal)

(1) Infantile eczema It is an eczematous condition on the face of children occurring any time from the age of two months and disappearing by the age of two year

Signs and symtoms Starts as erythemato vasicular lesions on face (Fig No 38 & 39) but scalp may be



Fig No 38
Infantile Eczema



Fig No 39
Infantile Eczema

involved and later on it may spread over the trunk Has a particular predilection for the face and flexural areas of the body Severe itching is present The

vesicles get ruptured and erythemato squamous leasions develop with exudation and itching The patient is a well nourished child and the nutrition does not suffer There are periods of quiescence followed by exacerbation The exacerbation occurs particularly at the time of teething

Diagnosis (1) Age of onset, (2) Well nourished child with eczema confined to face and flexural surfaces

Differential Diagnosis (1) Seborrhoic dermatitis, (2) Eczema in childhood, (3) Fungal infections

Prognosis Is good particularly with modern medicines But sudden death sometimes occurs Small pox vaccination should not be given

Treatment General—Antihistamins are given for 5 days by mouth such as Syrup Benadryl (P D), Antistin (Ciba) Phenergan (M & B) Change of brand of antihistamins from Antistin to Avil or some other may produce better results, Adrenocorticotrophic hormone (ACTH) may be used by injection but has a temporary effect Cortisone orally may be used but the result is temporary Hydrocortone ointment may be used locally but the result is not permanent

In the acute stage 1 p c aquous silver nitrate lotion as soaks are valuable but in the subacute stage Liniment calamine alone or with 2 p c Liq picis carb detergens may be used with benefit such as

Calamine ppt	dr 1
Olive oil	dr 4
Liq picis carb det	m 10
Aqua calcis	oz 1
Liniment for external use	

In the dry stage a tar ointment application with bandaging is necessary such as

Crude coal tar	dr 1
Zinc oxide	dr 1
Pulv amylum	dr 1
Vaseline alba	oz 1

Ung for external use

or Pragmatar or Primoderm may be used with benefit

Diet is most important in infantile eczema Milk and milk products should not be given Sometimes fruit juice is also contraindicated

Multivitamin and particularly vitamin C are given to the patient regularly Egg, lobster and crab are avoided in food

(2) **Besnier's prurigo** occurs after puberty usually in patients of infantile eczema and is characterised by erythematopapular lesions on the cubital and poplitial fossæ (Fig No 40) and sometimes on the forehead and sides of the neck

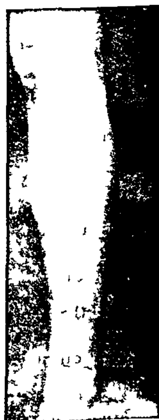


Fig No 40
Besnier s Prurigo

There may be acute exacerbation of the lesions followed by complete remission leaving only thickened skin in the cubital and popliteal spaces Itching is very severe and in acute stage there may be oozing

Treatment Change of environment is beneficial to help the psychological upsetting Patients are better treated in hospitals or nursing homes and should be kept away from relatives

Local treatment and general treatment are the same as for infantile eczema

X ray therapy is sometimes helpful

(3) **Nummular eczema** are erythematou, circular and coin shaped lesions on the extremities (Fig No 41) and are particularly on the exten or surfaces Itching is present and the skin is dry On the erythematous areas there is very slight sticky exudation present



Fig No 41
Nummular Eczema

Differential diagnosis (1) Psoriasis, (2) Contact eczema (3) Ringworm

Prognosis It is difficult to cure Relapses are common

Treatment General treatment consists of giving orally Vitamin A in high dose (100,000 i u) for a long time Locally when exudation is present 1 p c aquous gentian violet is painted followed by the application of 1 p c Ichthyol ointment and keeping the part bandaged up In the chronic stage tar is used as follows

Zinc oxide	dr 1
Pulv amylum	dr 1
Liq picis carb det	m 10
Vaseline alba	oz I

Ung for external use

Sometimes crude coal tar is helpful 10 p c crude coal tar in acetone is painted once daily and is dusted over with Pulv Zinc oxide and Pulv Amylum in equal quantity and then kept bandaged with a piece of linen smeared with an ointment containing

Zine oxide	dr I
Pulv amylum	dr I
Vaseline alba	oz 1

Ung for external use

X-ray therapy sometimes gives good result

(4) **Lichen simplex chronicus (Widal)** this is also known as neuro dermatitis Occurs in adults and particularly in those who shares heavy responsibility The lesions occur usually on the nape of the neck (Fig No 42), side of the neck, round about the albow, medial side of the thighs and front of the ankle Lesions are lozenge shaped papules which coalesce together to *form* a plaque which feels thick and tough and is pigmented at the periphery with criss cross markings Lesions

are very itchy (Fig No 43) Due to scratching strepto and staphylo get entrance through the scratch marks and produce eczematous reactions at times It persists from 3 months to 30 years or more Rarely it spreads and covers the whole body and is called **lichen simplex chronicus disseminatus**



Fig No 42
Lichen Simplex
Chronicus (Widal)

Prognosis Depends upon the psychology of the patient and proper handling of the case

Treatment Patient should be investigated psychologically and in resistant cases help of the psychiatrist is needed Phenobarbitone in grain 1 dose every night is helpful Largactil (M & B) 25 mg tab helps in dose of one tablet after food 3 to 4 times daily Locally when there is infection present it should be treated with 1 p c silver nitrate solution in water or 1 p c aquous gentian violet painting followed by the application of an ointment containing



Fig No 43

Lichen Simplex Chronicus (Widal)

1	Zinc oxide	dr 1
	Pulv amylum	dr 1
	Liq picis carb detergens	m. 10
	Vaselin alba	oz. 1
	Ung for external use twice daily	
or 2	Crude coal tar	dr 1
	Zinc oxide	dr 1
	Vaseline alba	oz 1
	Ung for external use once daily	

or 3 10 p c crude coal tar in acetone painted once daily or Pragmatar or Primoderm 1% may be used locally

4 Hydrocortone ointment may be used but the result is not permanent

5 "F99 ointment may be used with temporary benefit

ECZEMATID

Eczematid reaction occurs as a secondary rash in association with a pre existing eczema The toxin is absorbed from the site of the lesion and is toxic in nature Lesions are pinkish and pin head sized when appears at first but soon becomes oval in shape with a diameter of ½ to 1 inch and become itchy The lesions are vesicular and bullou at times and are particularly so when appear on the palm and sole Eczematid may be localized or generalised When generalised it is symmetrical in distribution

Id reaction when occurs in association with eczema and is due to bacteria it is known as 'bacterid, when due to fungus it is called trichophytid', when in association with tuberculosis it is called 'tuberculid, when in association with syphilis it is called 'syphilid' Pompholyx is supposed to be a special type of 'id reaction Some think that the origin of pompholyx is in the sweat duct

Differential diagnosis (1) Pityriasis rosea, (2) Seborrhoic dermatitis (3) Tinea corporis

Prognosis Is good

Treatment General treatment is to give an (1) alkaline mixture 4 times daily containing



Fig No 43

Lichen Simplex Chronicus (Vidal)

1	Zinc oxide	dr	1
	Pulv amylum	dr	1
	Liq picis carb detergens	m	10
	Vaselin alba	oz	1

Ung for external use twice daily

or 2	Crude coal tar	dr	1
	Zinc oxide	dr	1
	Vaseline alba	oz	1

Ung for external use once daily

DERMATITIS

Definition The skin disease which is characterized by the inflammation of the dermis and is associated with itching and erythemato papulo vesicular lesion with exudation and later crusting is called dermatitis

Classification (1) Drug dermatitis
(2) Artificial dermatitis
(3) Industrial dermatitis
(4) Seborrhoic dermatitis

DRUG DERMATITIS

Drug dermatitis is caused by the administration of one or more drugs

Etiology Hypersensitiveness to drugs is commonly seen in general practice and recently it has much increased due to the introduction of sulpha drugs, various antibiotics and continuous introduction of new drugs to the medical practitioners Sometimes a particular drug does not produce rash but when different drugs are used cross sensitization occurs and rash develops Sometimes inherited predisposition may be present

The predisposing factors to drugs are (1) increased susceptibility of the skin and (2) concentration of the drug in the skin

The increased susceptibility of the skin is due to the following factors such as

(a) Allergy which is a specific hypersensitiveness due to previous use of the drug The immunological mechanisms are responsible for this drug allergy

(1)	Pot cit	dr ½
	Liq ammon cit	dr 2
	Syp Aurantii	dr 1
	Aqua menth pip	ad oz 1
	Ft mist for a dose	

or Alkacitrone (Gluconate) 2 teaspoonfuls in water 4 to 6 times daily

(2) Antihistamin is given for 5 days in the form of Antistin (Ciba) as injection one ampule intramuscularly every night and one capsule of Benadryl in the morning and another capsule at noon for 5 days either alone or together with injection

(3) Vitamin C may be given orally in dose of 500 mg thrice daily or by injection once or twice daily

Locally may be used Lotio Calamine with phenol such as

Calamine ppt	dr 1
Phenol	dr ½
Aqua destil	oz 8 Lotio for external use

To be applied every hour on linen for 2 to 4 days and treat the eczema also

Lacto Calamine (Crookes), Caladryl (P D) or Calgesic (S & D) may be repeatedly used locally

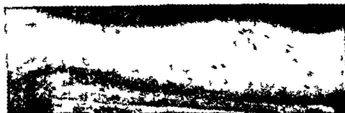


Fig No 44
Drug Rash
(Due to paludrine by mouth)



Fig No 45
Dermatitis Medicamentosa
(Due to [illegible])

One type of allergic response is seen in some drugs while several different types are seen with other drugs

(b) Idiosyncrasy is a peculiarity of an individual to develop drug rash even with therapeutic dose of a particular drug

(c) Photosensitization is the provocation of a drug rash with actinic rays of the sun

(d) Existing skin disease also helps to cause the development of drug rash

(e) Nervous irritability due to the toxic effect of the drug on the nervous system

The concentration of the drug in the skin as a result of (a) Preparatory to normal excretion—excretion is done through the sweat glands and sebaceous glands by exfoliation

(f) Impaired excretion by kidneys and bowels The mechanism of the production of the drug rash is not well known Many dermatologists consider the drug reaction to be an anaphylactic reaction

The drug hypersensitivity is generally a permanent affair *and the drug allergy is specific to a certain degree*

Drug rash is classified as follows —

(a) **Dermatitis medicamentosa** is caused by the administration of a drug either by mouth (Fig No 44) or by injection (Fig No 45) Examples are post-arsenical dermatitis (Fig No 46 & 47), gold dermatitis, bromide and iodide rash and the like

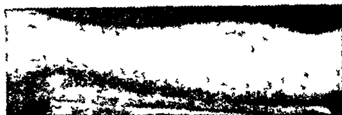


Fig. No 44

Drug Rash

(Due to paludrine by mouth)



Fig No 45

Dermatitis Medicamentosa

(Due to

One type of allergic response is seen in some drugs while several different types are seen with other drugs

(b) Idiosyncrasy is a peculiarity of an individual to develop drug rash even with therapeutic dose of a particular drug

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Fig No 48
Fixed Drug Rash
(Vesicles on erythematous base)

found in a patient The lips may also show pigmentation The skin lesion caused by a particular drug gets exaggerated following the admistration of the same drug The earliest lesion may be an itchy erythematous plaque with a sharp margin There may develop vesicles on the erythematous area which later undergoes involution leaving a pigmented macular lesion The pigmentation remains for a long time Drug rash is common in both the sexes and in all ages

Fig No 49
Fixed Drug Rash
(Due to Sulpha drug)





Fig No 46
Post arsenical Dermatitis
(Due to injection of arsenic)
(Case of Major S C P Sinha)



Fig No 47
Post arsenical Dermatitis
(Due to injection of arsenic)
(Case of Major S C P Sinha)

(b) **Dermatitis venenata** is caused by the local application of a drug on the skin Examples are sulpha drug rash, carbolic acid rash which develop after using them as dressings

(c) **Fixed drug rash** is a sharply circumscribed eruption which recurs in previously reacting areas after exposure to a drug (Fig No 48) The commonest variety of fixed drug rash is an erythemato squamous or hyperpigmented plaque Size varies from ½ to few inches in diameter (Fig No 49) In shape it is generally circular or oval One or several such plaques may be

Treatment Prophylactic is to remember that no dye, sulpha drug and antibiotic should be used locally

Before using a heavy metal or arsenic kidney function must be tested or the urine should be examined for the presence of albumin and casts The liver function test should be done when possible When facilities are available blood vitamin C estimation is done because vitamin C is the normal physiological detoxicating agent in the body Patients with low blood vitamin C usually develop drug rash

Curative treatment consists of (1) stopping the drug, (2) One dose of S S Mag sulph is given by mouth, (3) 25 p c glucose solution 25 c c to 50 c c is injected intravenously with or without vitamin C (500 mg) for two weeks (4) Vitamin C (500 mg) is given twice daily by intramuscular injection or thrice daily by mouth in tablet form (500 mg) for a week or 10 days, (5) Sometimes BAL or Sodium thiosulphate (Ametox sodium—May and Baker or Contramine—British Drug House) may be used by injection for 5 to 7 days (6) When the drug rash is very severe in nature ACTH aquous solution is injected 6 hourly in 40 units dose for 3 days and then ACTH gel once daily for the rest of the week Cortisone (25 mg) tablet may be used orally every 6 hours for 3 days 15 mg for 3 days 10 mg for 3 days and 5 mg for 3 days and afterwards 5 mg twice daily for a week During the corticotropine or cortisone therapy the patient should be without salt and must have vitamin B complex 4 times daily by mouth

Locally in the early stage Lotio calamine is applied every hour Sometimes Liniment calamine is helpful

Signs and symptoms Sometimes the rash is localized to a particular region of the body while generalised rash may also occur The rash may be a temporary erythemato macular or papular lesion Erythema multiforme type of rash and urticarial may also occur Bullæ can also be seen and even granulomatous lesions may develop in chronic poisoning Hyperkeratosis of palms and soles may occur Gangrene may also occur Epithelioma has resulted as a complication in arsenic therapy

Sometimes the rash develops within four hours after the administration of the drug such as in quinine, sulpha drug and penicillin rash whereas at other time the drug rash appears after a long time from a few days to a few weeks after the administration of a drug As a rule the drug rash appears suddenly and disappears suddenly after the stoppage of the drug

Diagnosis (1) Sudden appearance of the rash, (2) Distribution of the rash being symmetrical, (3) Character of the rash—polymorphic, (4) History of the patient of drug taking, (5) Patch test is sometimes helpful, (6) Biopsy—histopathology—In a fixed drug rash the stratum basalis will show increased melanin formation in the melanoblast cells and the presence of melanin laden chromatophores may be found throughout the dermis The papillary blood vessels are found dilated with perivascular infiltration with lymphocytes and histiocytes In the granulomatous bromide rash known as bromoderma pseudo epitheliomatous hyperplasia of the epidermis is seen with multiple micro abscesses

Prognosis Good with modern drug like ACTH and cortisone

Signs and symptoms Found on the body which is within easy reach of the patient's hands Legs (Fig No 50) and arms and forearms (Fig No 51) are common sites Lesions are mostly found on the anterior surface of the body and on the face (Fig No 52) Lesions have sharp margins



Fig No 51
Dermatitis Artifacta



Fig No 50
Artificial Dermatitis
(Below the left Knee)



Fig No 52
Dermatitis Artifacta

with or without 1 p c phenol An ointment may be used containing

Calamine ppt	dr	1
Menthol	gr	10
Pulv Amylum	dr	1
Vaselin Alba	oz	1

Ung for external use

Later on olive oil with or without 1 p c Hydrarg Ammon or 1 p c phenol may be used When there is oozing 1 p c Silver nitrate soaks are applied every hour for 2 or 3 days When exudation has almost subsided 1 p c aquous gentian violet lotion is painted followed by the application of 1 p c ung Ichthyol In chronic stage it is treated as chronic eczema

Sometimes in very severe non oozing type of drug rash Hydrocortone ointment locally may be used with benefit

ARTIFICIAL DERMATITIS

This is also known as Dermatitis Artefacta, Dermatitis Autophytica, Dermatitis Factitia, Self-inflicted Erruptions, Feigned Dermatitis or Hysterical Dermatitis

Definition Artificial dermatitis is characterized by erythema, ulceration and even gangrene caused by the neurotic patients on their own bodies

Etiology Beggers usually produce ulcers on their bodies, on the arms or legs to rouse sympathy and the malingerers do it to avoid *doing any work* *Patients* are mostly females Persons of any age may have the disease but is common amongst young women

(b)	Liniment calamine	Calamine ppt	dr 1
		Oil Olive	dr 4
		Aqua calcis	ad oz 1
		Liniment for external use	
(c)	Ointment	Calamine ppt	dr 1
		Acid salicylic	gr 10
		Vaseline alba	oz 1
		Ung for external use	

Permanent dressings when left for 3 to 6 days with an ointment helps to heal the lesions

(d)	Ointment	Calamine ppt	dr 1
		Pulv amylum	dr 1
		Liq Picis carb det	m 10
		Vaseline alba	oz 1
		Ung for external use	

Sometimes these patients are very clever and intelligent and it becomes difficult to treat them at their places of residence It has often been found helpful to treat these patients in hospital under efficient nurses

INDUSTRIAL DERMATITIS

Also known as occupational dermatitis or trade eczema

Definition Industrial dermatitis is caused by the application of different chemicals application of heat and due to mechanical causes such as by dust or friction to the skin while at work in an industry producing eczematous reaction

Etiology One per cent of workers usually suffers from industrial dermatitis (Schwartz) In the tropics the

Diagnosis (1) The stigmata of sensitiveness must be sought for such as nail biting facial expression and the mental make up are important factors, (2) Sites of lesions, (3) Absence of conjunctival and faucial reflexes, (4) Hypo sensitiveness to pain near the site of lesion, (5) Hyperaesthesia is a common symptom, (6) Patient often predicts the sites of fresh lesions on the skin

Prognosis Complete cure is the rule

Treatment Prophylaxis psychosomatic skin diseases have started receiving attention and a skin specialist should try to look to a patient from that angle also

Curative—the environmental relationship with the patient should be carefully studied and the patient's socio economic and domestic situations must be taken into account and hospitalization of the patient is helpful Calming functional irritability is important which may be done by psychotherapy and by good understanding between the doctor and the patient The doctor need be very sympathetic Sedatives are helpful and as a routine *Elixir Bromo Valerian dr 1 with Elixir Vitamin* B Complex dr 1 may be given after food 3 times daily for about 2 months Largactil (M & B) is helpful Sometimes Valerian (B C P W) is injected intramuscularly biweekly Phenobarbitone is very helpful as a sedative when used for a long time Locally may be used

(a) Lotio calamine—Calamine ppt dr 1
Zinc oxide dr 1
Glycerine pure dr ½
Aqua calcis ad oz 1
Lotio for external use

industry Sometimes the patch test is negative to one particular substance but is positive to a mixture of two or more substances used in the industry

Prognosis Is good with treatment and with proper preventive measures Sometimes a change in occupation is needed for cure

Treatment Prophylaxis (a) Proper selection of the personale in the industry, (b) washing of hands and feet immediately after handling any irritant or using over alls and gloves (c) Facilities to change clothings, bathing and proper ventilation of the factory are essential In tropical countries putting on electric fans or air conditioning of factories may help in checking of various industrial dermatitis, (d) Educating the industrial workers by lectures, cinematography etc to avoid the industrial dermatitis should be instituted, (e) Barrier creams should be used to protect skin

General—Rest with treatment The skin lesion is treated as any eczema

If it is in the acute eczematous stage repeated local applications of lotio calamine is the best treatment When the condition is in the sub acute stage with oozing 1 p c lotio silver nitrate soaks are applied every hour for a day or two and then the oozing almost disappears In this stage 1 p c lotio gentian violet application once daily followed by the application of 1 p c ung Ichthyol and keeping the part bandaged repeating this daily for a week In the chronic stage a bland ointment with 2 p c Acid salicylic or 2 p c liq picis carb det or with both is applied daily and kept bandaged repeating this for abou a week or so

number of cases are increasing with the growth of industry in the country The irritants enter the body through the hair follicles and sweat ducts but the fat soluble *chemicals get* absorbed through the skin Irritation produced on the skin either by the contact of chemical agents, physical agents or by mechanical means produces dermatitis which is designated as the industrial dermatitis Workers may suffer in any industry Even *the typist who is handling the type tape or carbon* paper suffers The printer who handles different chemicals, the mason's skin is traumatized by sand and cement, the miner's skin also gets traumatized by coal dust Predisposing factors are many such as (1) history of allergy such as eczema, urticaria, asthma, drug idiosyncrasy and (2) oily skin and seborrhoic diathesis

Age – may occur at any age
Sex—may occur in both sexes

Signs and symptoms Erythematous lesion is the earliest sign and itching of hands and body may be the earliest symptom There may be erythemato papular, papulo vesicular or pompholyx like lesions, ulceration and gangrene may occur Rash may cover the whole body There may be intractable itching Scratching may be followed by oozing and then crusting Cellulitis may occur with edema of face, hands and feet There may be albuminuria and microscopic haematuria (For pictures see eczema due to contact on page 40)

Diagnosis (1) Worker in an industry, (2) Skin lesions on exposed parts of the body usually but may be over the whole body, (3) The skin lesion spreading far beyond the area of contact with the material in the industry, (4) Skin test with the material used in the

Urticaria is an allergic reaction due to the introduction of a foreign protein in the body of a sensitized individual (d) Psychological disturbance may cause urticaria (e) Hodgkin s disease leukaemia and reticulo sarcomas may be preceeded by urticaria

Classification (a) Urticaria papulosa
(b) Urticaria bullosa
(c) Urticaria gyrata
(d) Urticaria haemorrhagica
(e) Urticaria factitia or dermographism
(f) Angioneurotic edema or Quincke s syndrome
(g) Familial urticaria
(h) Urticaria pigmentosum

Signs and Symptoms Sudden development of wheals with intense itching which is **Urticaria papulosa** (Fig No 53) The wheal may be bullous when it called **Urticaria bullosa** When the lesions are very large and of policyclic in shape it is known as **Urticaria gyrata** Sometimes there are haemorrhagic plaques in the



Fig No 53
Urticaria Papulosa
(Case of Dr B N Banerji)

TOXIC DERMATOSES

Toxic dermatoses are due to unknown toxins

Classification (1) Urticaria
(2) Prurigo
(3) Purpura
(4) Erythema multiforme
(5) Erythema nodosum
(6) Dermatitis herpetiformis
(7) Pemphigus
(8) Epidemic dropsy

URTICARIA—Definition Is an acute or chronic disorder of the skin characterized by the development of wheals on the body with intense itching

Etiology Urticaria is due to allergic reactions In an acute case of urticaria a positive skin test can be found whereas in a *chronic case the skin test (Prausentz—*Kustner reaction) is usually negative Urticaria may be due to (a) external causes such as due to heat, light and *cold It is said that the patients* have an antibody in their serum which reacts with the physical agents on exposure of the skin and develop urticaria The stings of insects are the common causes of urticaria (b) Internal causes such as are often due to the ingestion of some drug like aspirin, sulphonamide, injection of antibiotics and the like, (c) Parasitic causes such as in the tropics are intestinal infections like round worm, amaebiasis, giardiasis and *the like and in malaria infection* Percival thinks that to precipitate an attack of urticaria the state of the tissues are more important than the noxious *substance* which is protein in nature

The lesions are commonly pink coloured papules which suddenly appear and disappear in about 3 hours' time Gradually the lesions disappear at longer intervals and in chronic cases it becomes persistent This may be acute or chronic Acute urticaria may be accompanied



Fig No 55
Angioneurotic Edema
(Case of Dr K C Kandhari)

with intense itching redness, nausea and vomiting Chronic urticaria is a recurrent condition Dermographism may not be found in patients with urticaria

Diagnosis (1) Sudden appearance of pinkish papular lesions over the skin with itching which disappears after about 3 hours in acute cases but in chronic cases it is persistent (2) History of indiscretion to diet or administration of drug or serum injection, (3) Histopathology Urticaria is a triple response to the effect of histamin like substance produced by the tissue

wheals or separate from it, this is a rare type and goes by the name of **Urticaria haemorrhagica** In some individuals the skin is so irritable that slight scratching causes wheals to develop when it is called **Urticaria**



Fig No 54
Dermographism
(Case of Dr K C Kandhari)

factitia or **Dermographism** (Fig No 54) When large swellings appear on different parts of the body and involve the eyelid or lip it is called **Angioneurotic edema** (Fig No 55) **Familial Urticaria** is found in several members of the same family and is often due to psychological upset **Urticaria pigmentosa** is a chronic skin disease which starts within the first year of life and is characterized by urticarial pigmented macules for which see page 34

(Winthrop) after food 3 times daily for 10 days but there are other oral preparations available in the market which have effect both on intestinal and extra intestinal parasitic infections During this ten day therapy the pH of the gastro intestinal tract is changed by giving a special diet consisting of Rice, Dahi (Butter milk) and boiled vegetables followed by normal diet with high protein Similarly for giardiasis atebrin tablets are given for 5 days in dose of one tablet after food 3 times daily

The blood of the patient is examined for total count of W B C R B C, Differential count, Haemoglobin per cent and for parasites If malarial parasites are found the patient is treated accordingly

Saline purgative like S S Mag Sulph Oz 1 is usually given in the acute cases but in chronic cases Mist Alba Oz 1 thrice daily for 3 to 7 days has been found more efficacious

Autohaemotherapy is helpful starting with 2 5 c c of blood and increasing by 0 5 c c upto 5 0 c c are injected biweekly I M for 6 to 12 such

Antihistamine drug is given in acute stage by daily intramuscular injection for 5 days but in chronic cases 50 mg orally thrice daily for 5 days is found to be effective When after 5 days' therapy there is no response it is helpful to change the brand of the antihistamines Antihistamine drugs do not neutralise all the histamines produced in the body They act by blocking the receptors of histamine Urticarial wheals can be reduced or suppressed by antihistamine drugs and the underlying cause of wheal formation is not affected All the antihistamine drugs have the same essential action

damage Edema in the dermis in all the tissues with dilatation of papillary vessels In chronic cases there is found cellular infiltration with leucocytes and mast cells

Differential Diagnosis

(1) Prurigo nodularis (2) Drug rash
(3) Secondary syphilis (4) Hodgkin's disease
(5) Leukaemia cutis (6) Mycosis fungoides

Prognosis Is good in acute urticaria In chronic urticaria patients suffer for a long time Angioneurotic edema causes swelling of eyelids, lips and even trachea producing breathing difficulty and may even cause death

Treatment Prophylaxis—In the tropics amaebiasis and giardiasis should be avoided by observing scrupulous cleanliness of cooking vessels and utensils and stool should be examined at regular intervals and treatment is taken when needed for intestinal infection In a malarial place mosquito curtains should be used and prophylactic anti malarial therapy should be given All foci of infection should be investigated and should be treated Patient should be investigated from the psychological point of view and rehabilitation of the patient is necessary as a prophylactic measure

Curative—In the tropics the patient should be thoroughly investigated for gastrointestinal parasitic infections like giardiasis, amaebiasis, blantidiasis and the like and should be treated accordingly Chronic amaebiasis is very intractable and the routine treatment should consist of (1) Six daily intramuscular injection of emetine hydrochloride gr 1 with rest together with (2) one tablet of Entero vioform (Ciba) or Entero quinol (East India Pharma Works), Siostearn (Geigy) or Aralis

Quinine bihydrochlor gr 5, thrice daily, for a week is of value in urticaria associated with malaria in the tropics Colonic lavage with Condy s lotion (1 in 1000) or with normal saline is helpful in chronic urticaria and angioneurotic edema

Moccasin venom in dose of 0 1 c c (1 300 000 solution) at 7 day intervals and increasing by 0 1 c c upto 1 0 c c has been found beneficial in chronic urticaria of unknown cause

Locally warm water bath twice daily gives relief Bath may be followed by application of a powder consisting of

Pulv Camphor	dr	½
Pulv Zinc oxide	dr	1
Pearl Powder (Bengal Chemical)	oz	1
Ft Pulv for external use		

Lotion may also be used such as

(1)	Calamine ppt	dr	½
	Liq Picis Carb det	m	10
	Aqua Distil	oz	1
	Ft Lotio for external use		
(2)	Calamine ppt	dr	½
	Phenol	m	5
	Aqua Distil	oz	1
	Ft lotio for external use		
(3)	Calamine ppt	dr	½
	Zinc oxide	dr	½
	Phenol	m	5
	Oil olive	dr	½
	Aqua calcis	oz	1
	Ft liniment for external use		

on urticaria Antistin tablet may be given as one tablet thrice daily or Phenergan Benadryl, Anthisan, Dibistin may also be used orally Calciluvin (Bœhringer) by injection may be given I M daily Benadryl or Antistin may be used as injection as one ampule daily for 5 to 7 days

Sometimes the antihistamine drug may itself act as an allergen and produces shock which is called "histaminoid accidents" This reaction may occur during or after the treatment

Cortisone may be given in 10 mg dose every 6 hours Cortisone causes rapid disappearance of the urticarial wheals and itching It should not be used in acute urticaria where antihistamines can safely be used For chronic urticaria cortisone may carefully be used when it is difficult to control the intractable urticaria

Vitamin C (500 mg) by intramuscular injection every 12 hours or by mouth in tablet form every 6 hours for a week are helpful in chronic cases Sometimes a combination of Calcium with Vitamin C is helpful

Ephedrine sulphate in 25 mg dose may be given orally once or thrice daily in urticaria and may be repeated 3 to 4 times in angioneurotic edema

Adrenaline *hydrochlor (1 in 1000) by intramuscular* injection in dose of 0 5 to 1 0 c c is particularly helpful *in acute* and severe attacks of the disease

Thyroid extract gr ¼ tablet daily for a week is sometimes *helpful in chronic urticaria*

Ekzebrol (Tosse) when injected intravenously in gradually increasing doses helps to control the itching in urticaria and has a sedative effect also

(3) Antihistamine ointments are sometimes used for antipruritic effects But epidermal sensitization often occurs by the topical use of antihistamines in the form of ointment Some dermatologists have found the combination of antihistamine orally and locally to be more effective in giving relief to the intense itching of urticaria as the antihistamines act by their central analgesic and local anaesthetic effect Neuropsychiatric investigation is needed when nothing helps the patient

Psychological investigation to find out the precipitating factors and psychotherapy are helpful in some intractable cases of urticaria

Diet Is important in urticaria The taking of egg or drinking of milk or some other such foods sometimes cause urticaria and it is helpful to avoid such things during disease Elimination diet is helpful During the treatment the patient should be put on bland and lacto vegetarian diet Sometimes milk is also stopped and the patient is put on rice or wheat products and vegetables for sometimes and then is gradually taken to normal diet by adding one article every third day

PRURIGO

Definition Is a chronic skin disease characterized by itching and nodule formation, some of which are excoriated and are found generally on the limbs

Etiology Cause is not known This is associated in the tropics with malnutrition and psychological upset Sex found in both sexes Age in people of past middle age but there is often a history of its onset early in life

(4)	Calamine ppt	dr ½
	Zinc oxide	dr ½
	Menthol	gr 2
	Glycerine pure	dr 1
	Aqua calcis	oz 1
	Ft lotio for external use	

(5)	Thymol	gr *2*
	Glycerine	m *20*
	Aqua calcis	oz 1
	Ft lotio for external use	

(6)	Lotio Nigra	dr *3*
	Oil Olive	dr 3
	Aqua calcis	ad oz 1
	Ft liniment for external use	

(7) Caladryl (Parke Davis) Calmitol lotion (Siegfried), Calgesic ointment (Sharpe & Dhome) when applied locally has soothing effect in urticaria

Ointment may be used such as

(1)	Calamine ppt	dr ½
	Zinc oxide	dr ½
	Menthol	gr 5
	Vaseline *Alba*	ad oz *1*
	Ft Ung for external use	

(2)	Zinc oxide	dr ½
	Camphor Pulv	gr 12
	Menthol	gr 3
	Oil Eucaliptol	m 2
	Vaseline alba	ad oz 1
	Ft Ung for external use	

extensor aspects of the extermities (Fig No 57) but may also be found on the trunk over the suprascapular



Fig No 57
Prurigo nodularis

regions (Fig No 58) Starts late in life but usually after puberty and persists throughout life Patients cannot have



Fig Fo 58
Prurigo nodularis

Types (1) Prurigo simplex
(2) Prurigo nodularis

Signs and symptoms **Prurigo simplex** is quite common and are found in all ages and in both sexes in the tropics These are characterized by papular lesions on the limbs, particularly over the gluteal regions, arms and thighs Lesions are skin coloured and are itchy Itching causes excoriation and relief Starts early in life before puberty and rarely disappears in adult age Prurigo simplex rarely becomes prurigo nodularis in adult age **Prurigo nodularis** is not uncommon in the tropics Is often found in elderly males Lesion is nodular and in size that of a pea (Fig No 56) It is of skin colour and is very very itchy Generally distributed over the arms and thighs (Fig No 56),



Fig No 56
Prurigo nodularis

Curative Antihistamine may be tried in dose of 25 mg tablet 3 to 4 times daily for a week and then with a maintenance dose of 5 mg thrice daily for weeks Phenobarbitone may sometimes be needed orally in dose of gr ¼ to ½ once or twice daily Ekzebrol (Tosse) intravenously is helpful in some case

Locally (a) antipruritic lotions may be used containing

(1) Calamine ppt	dr	1
Phenol	gr	10
Aqua distil	oz	1
Ft Lotio for external use		
(2) Calamine ppt	dr	½
Sulpher ppt	gr	10
Liq Picis Carb det	m	10
Aqua Distil	oz	1
Ft Lotio for external use		

(3) Antihistamine lotions may be used but sensitization may develop sooner or later with it (4) Caladryl (P D) and Calmitol (Siegfried) give temporary relief from itching

(b) Ointments may be used in from of

(1) Acid Salicylic	gr	10
Liq Picis Carb det	m	10
Vaseline Alba	oz	1
Ft Ung for external use		

(2) Antihistamine ointments may be used but there is a risk of sensitizing the patient

good sleep and are so such mentally disturbed by constant itching that they develop suicidal tendency also

Diagnosis (1) Nodular itchy and excoriated lesions distributed over the extremities and on the back, (2) Nervous type of patient, (3) In children bed itching is a common symptom, (4) Histopathology hypertrophy of *stratum corneum stratum granulosum* and *stratum mucosum* is seen in prurigo nodularis (Fig No 59) while in prurigo simplex there is dystrophy of the dermis

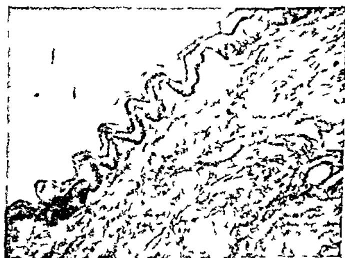


Fig No 59
Histopathology of Prurigo nodularis

Differential Diagnosis (1) Urticaria, (2) Drug Rash, (3) Scabies, (4) Pediculosis, (5) Lichen planus nodularis, ( ) Secondary syphilis, (7) *Von Recklinghausen's disease*

Prognosis Persists throughout life

Treatment Prophylaxis consists in removal of the patient to a better *surrounding and away* from psychological and familial influences

Curative Antihistamine may be tried in dose of 25 mg tablet 3 to 4 times daily for a week and then with a maintenance dose of 5 mg thrice daily for weeks Phenobarbitone may sometimes be needed orally in dose of gr ¼ to ½ once or twice daily El zebrol (Tosse) intravenously is helpful in some cases

Locally (a) antipruritic lotions may be used containing

(1) Calamine ppt dr 1
Phenol gr 10
Aqua distil oz 1
Ft Lotio for external use

(2) Calamine ppt dr ½
Sulpher ppt gr 10
Liq Picis Carb det m 10
Aqua Distil oz 1
Ft Lotio for external use

(3) Antihistamine lotions may be used but sensitization may develop sooner or later with it (4) Caladryl (P D) and Calmitol (Siegfried) give temporary relief from itching

(b) Ointments may be used in from of

(1) Acid Salicylic gr 10
Liq Picis Carb det m 10
Vaseline Alba oz 1
Ft Ung for external use

(2) Antihistamine ointments may be used but there is a risk of sensitizing the patient

good sleep and are so such mentally disturbed by constant itching that they develop suicidal tendency also

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Fig No 59
Histopathology of *Prurigo nodularis*

Differential Diagnosis (1) Urticaria, (2) Drug Rash, (3) Scabies, (4) Pediculosis, (5) Lichen planus nodularis, ( ) Secondery syphilis, (7) Von Recklinghausen's disease

Prognosis Persists throughout life

Treatment Prophylaxis consists in removal *of the* patient to a better surrounding and away from psychological and familial influences

Prognosis Good

Treatment Prophylaxis consists of taking balanced deit and avoiding infectious disease

Curative Rest in bed Vitamin C (500 mg) intra muscular injection every 6 hours for 3 days and then twice daily Sometimes repeated blood transfusion is required Vitamin C with Calcium is also helpful

Sometimes antibiotic therapy is indicated such as crystalline Penicillin 'G' 5 lacs (0 5 Mega unit) intra muscularly in water every day for 5 days

ERYTHEMA MULTIFORME

Definition Is characterized by the recurrent occur rence of erythemato papular lesions on the body

Etiology (1) Virus is said to be responsible for certain types of erythema multiforme such as Stevens Johnson syndrome (2) Herpes simplex virus is said to be responsible, (3) Virus pneumonia may be respon-sible, (4) Toxin is responsible for other types of the di eases such as erythema iris This toxin may be bacterial or due to a drug, (5) Endocrine diseases are somtimes responsible

Age—Commonly affects children Seen in both sexes

Classification (1) Idiopathic type
(2) Multiforme type
(3) Stevens Johnson type

Signs and Symptoms In the idiopathic type the lesions are erythemato papular and the situated on the

Surgical treament is helpful 1 p c local infiltration with novocaine of the skin arond a nodule is anaesthetised and the nodule is excised and stitched One or two such nodules may be taken out weekly

X ray therapy is not of much use in these patients

PURPURA

Definition Purpura is charactarized by haemorrhage in the dermis with itching

Etiology Types—(1) Idiopathic
(2) Symptomatic

Age—at any age Sex—may be found in both sexes

Signs and Symptoms Idiopathic purpura is characterized by fever, gastro intestinal disturbance, swelling of joints and haemorrhage in the skin In symptomatic purpura there is also haemorrhage in the skin in association with other diseases such as herpes zoster, pemphigus, infectious fevers, during the toxaemic stage and Vitamin C dificiency stage Recurrences are common Sometimes only the ankles may be affected with lichenoid purpuric pigmentation without any blood change except showing sub clinical avitaminosis C in adult males which is called pigmented purpuric lichenoid dermatitis

Diagnosss (1) Haemorrhagic patches in the skin of the body and are particularly confined to the ankles, (2) No blanching of the lesion on diascopy examination, (3) Examination of blood for total count of W B C and R B C, differential count, hæmoglobin per cent, parasite and platelet count, (4) Histopathology shows deposits of haemosiderin in the dermis

conjunctiva (Fig No 61) There may be rhinitis urethritis and vaginitis Is common in both sexes and

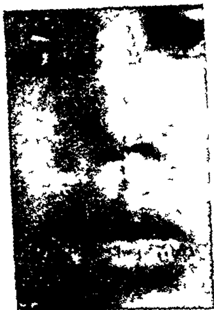


Fig No 61
Erythema multiforme
(Showing lesions on lips and eyes)
(Patient Meena Kumar)

can be seen in all ages Skin rash may appear several days after the illness

(b) Bahcet s syndrome is characterized by inflammation and erythema of the mucous membrane of *conjunctiva*, mouth and genitalia with fever There may be intense photophobia and even Keratitis with circumorbital pain Oral lesions are erthymatous with a red *halo* There may be pain in the tonsils and in the tongue Common amongst children and is found in both sexes

(c) Reiter s syndrome is characterized by conjunctivitis, urethritis and arthritis Seen in adults Found in both sexes but common amongst females Skin lesions are rarely seen when present the skin lesions are urticarial erythematous or nodular in nature The

extremities and even on palms and soles Some of the lesions are circular and are called **Erythema iris** Sometimes bullæ are found In the multiforme type the lesions are large in size and are characterized by erythematopapular lesions covering *the trunk or the extensor surfaces* of the limbs (Fig No 60) Distribution is symmetrical There may be ulceration of mouth with or without any rash on the body Onset is rapid

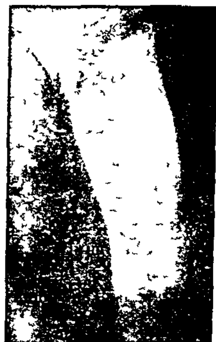


Fig No 60
Erythema multiforme
(Erythemato papular lesion on thigh)

The Stevens Johnson type is characterized by fever with inflammation of the mucous membranes of mouth and conjunctiva There are three varieties of Stevens-Johnson syndrome such as

(a) Stevens Johnson syndrome is also called Ectodermosis pluriorificialis erosiva where there is erythema and ulceration of the mucous membrane of mouth and

presents a relapse, (7) ACTH injection is sometimes helpful at 6 hours, 8 hours, 12 hours, intervals of 40 units of the aqueous solution and then ACTH gel 40 units at 24 hours interval and subsequently reducing the dose to 30 units, 25 units, 20 units and 10 units, (8) Antihistamines are sometimes helpful in a dose of 50 mg at 6 hours interval for 5 days, (9) Hydrocortisone ointment (Roussel) locally allays itching

Diet Bland diet consisting of liquids like milk, tea, fruit juice for a week then fish and meat can be added with bread and butter If cortisone is used salt free diet should be given or K salt (Calcutta Chemical) may be added to food

ERYTHEMA NODOSUM

Definition Is a skin disease characterized by the development of symmetrical painful nodules on the legs below the knees with slight fever

Etiology (1) Tuberculosis may be the cause and may be the sign of activation of a latent focus (2) Rheumatic origin is also suspected, (3) Pyogenic infection—may be associated with strepto and staphylo coccal infection of tonsils and sinuses (4) Meningo coccus—erythema nodosum may be symptomatic of meningococcal infection of the cerebro spinal system, (5) Gonococci sometimes erythema nodosum may develop in association with gonococcal septicæmia (6) Leprosy—it may develop in a patient suffering from lepromatous leprosy (7) Syphilis—erythema nodosum has often been found in syphilitics (8) Drugs—erythema nodosum has often developed during or after therapy with certain

eruptions are found on hands, feet and genitals The disease runs a long course of 4 to 6 months

Diagnosis (1) Recurrent occurrence of erythmato papular lesion which are symmetrical and are mostly on the extensor surfaces of the limbs, palms, soles with or without fever and conjunctivitis, (2) Histo pathology shows edema of the whole dermis including the vessels and lymphatics with dilatation of blood vessels and lymphatics Round cell infiltration which are mostly eosinophilic in the early stage but later on changing to lymphocytes In the erythema multiform bullosum type the situation of the bulla is intra epidermal

Differential Diagnosis (1) Uurticaria (2) Dermatitis herpetiformis, (3) Drug rash, (4) Pemphigus

Prognosis Is good in most cases The eruptions subside in 2 to 3 weeks

Treatment Prophylaxis—not know yet

Curative—(1) Rest in bed (2) Saline purgative (3) Alkali mixture 4 times a day with or without Sodi Salicylas - gr 10 per dose

(4) Aureomycin capsule (250 mg) orally every 6 hours for 4 days together with Elixir Vitamin B Complex, 2 tea spoonful twice daily are helpful

(5) Vitamin C (500 mg) is injected intramuscularly twice daily for 7 days together with 10 p c calcium gluconate solution, (6) Cortisone (25 mg) by mouth every 6 hours with vitamine B complex for 4 days and then gradually decreasing the dose before finally stopping the drug shortens the duration of the attack but never

presents a relapse (7) ACTH injection is sometimes helpful at 6 hours, 8 hours, 12 hours intervals of 40 units of the aquous solution and then ACTH gel 40 units at 24 hours interval and subsequently reducing the dose to 30 units, 25 units, 20 units and 10 units, (8) Antihistamines are sometimes helpful in a dose of 50 mg at 6 hours interval for 5 days (9) Hydrocortisone ointment (Roussel) locally allays itching

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drugs like sulpha drugs, antibiotics, bromides, iodides and the like, (9) Sarcoidosis may be associated with it, (10) Ulcerative colitis may give rise to erythema *nodosum*

Sex-may affect both sexes Age may affect any age

Signs and symptoms Malaise or slight fever may be present for a day or two before the development of painful, dusky red nodule half pea sized to four times the diameter of a pea The lesions *develop on the calf muscles and on the front of the* legs below the knee joints Lesions are tender and occur on both the legs They are painful Nodules never suppurate nor ulcerate In 2 to 4 weeks time lesions gradually fade away

Diagnosis (1) Symmetrical dusky red, tender and painful nodules on both legs below the knees, (2) No *tendency to ulcerate,* (3) Blood W R and Khan tests are negative except in cases associated with syphilis, (4) Tuberculin test (Mantoux test) is possitive, (5) E S R is high, (6) Biopsy—histopathology shows edema in the dermis with dilatation of blood vessels and infiltration with round cells and epitheloid cells Rarely giant cells are seen

Differential diagnosis (1) *Drug rash,* (2) Gumma, (3) Erythema multiforme

Prognosis *Is good Seldom there is a relapse*

Treatment (1) Rest in bed is very important, (2) Skiagram of chest to find out a tuberculous focus

for treatment (3) Legs are wrapped with cotton wool and kept at rest (4) A mixture is given containing

Sodii Salicylas	gr 10
Sodii Bicarb	gr 15
Liq Ammon Cit	dr 1
Syp Calcii hypo	dr 1
Aqua menth pip	ad oz 1

Mft Mist for a dose Send 8 such Is taken every 6 hours (5) A saline purgative is given at the beginning of the treatment (6) Sulpha drug such as sulphadiazine tablet every 6 hours for 4 days may be given, (7) Penicillin crystalline G' in aquous solution of 5 lacs daily is helpful (8) Vitamin C (500 mg) may be injected at 12 hours interval or vitamin C (250 mg) tablet is given by mouth every 6 hours for a week

DERMATITIS HERPETIFORMIS

Definition Is a bullous dermatitis characterised by grouping itching pigmentation and recurrences

Etiology It is seen in the tropics and form about 1 0 per cent of all skin cases Sex—occurs in both sexes Age—found in all ages but is common amongst children in the tropics

Cause is not known but some toxin is supposed to be responsible for its causation Some think that a virus is resposible for the development of dermatitis herpetiformis Pregnancy is a predisposing factor and the dermatitis herpetiformis which develops during pregnancy is known as **herpes gestationis** or hydroa gravidarum **Impetigo herpetiformis** is another grave and rare form of dermatitis herpetiformis which affects males and the females

Signs and symptoms Eruptions are erythematous or bullous in type Bullæ develop for several days with burning and itching

They are grouped and hence known as herpetiformis Lesions are symmetrical

There is no erythematous halo round the vesicle

Sites are sacral region, buttocks, round the elbows, knees and scapular regions (Fig No 62 & 63 In male children sometimes is seen on the genitalia Itching is felt long before the eruption appears on the skin and itching is relieved after the rupture of the bullæ When the ulcer (Fig No 63) heals pigmentation is left Rarely the



Fig No 62
Dermatitis herpetiformis



Fig No 63
Dermatitis herpetifor

mucous membrane is also involved Herpes gestationis develops early in pregnancy between the third and sixth month Lesions are erythematous papules or vesicles and are found in groups (Fig No 64) distributed on the



Fig No 64
Herpes gestationis

forearms, legs, buttocks, back and abdomen (Fig No 65) Eruptions disappear after the delivery The child is



Fig No 65
Herpes gestationis

not affected by the eruptions but stillbirths and miscarriages may often occur This is a recurrent condition

Diagnosis (1) Symmetrical recurrent itchy grouped bullous lesions on sacral region and round the elbows and knees (2) Multiformity of lesions, (3) Chronicity, (4) Pigmentation (5) Eosinophilia in blood which may be above 30 p c and the variability of the eosinophil cells in blood on different days is peculiar in dermatitis herpetiformis, (6) Cytology of a bulla will show over 30 p c of eosinophil cells (7) Biopsy—histopathology will show a subepidermal bulla and the presence of œdema in the dermis and epidermis Perivascular dense cell infiltration eos nophil in the dermis Hyper pigmentation below the stratum basalis is common

Differential diagnosis (1) Urticaria (2) Scabies, (3) Impetigo (4) Erythema multiforme (5) Congenital bullous syphilitic rash, (6) Drug rash (7) Pemphigus

Prognosis Is good so far as the life is concerned in the younger age group There are always relapses of this condition and in particular during pregnancy In old age dermatitis herpetiformis may change over to pemphigus when prognosis becomes grave Herpes gestationis does not affect pregnancy and gets well after termination of pregnancy

Treatment Prophylaxis investigation of all septic foci and their treatment are essential Patients develop ing herpes gestationis should avoid further pregnancies Rh factor of parents may be responsible for the develop ment of the disease in children

Curative—All hygienic measures should be adopted In the tropics cold water bath every day and warm water bath in winter is essential Bland soap may be used for bath

Arsenic is an old drug for this disease and is used in the form of liquor potasii arsenitis (Fowler's solution) given for a long time starting with one drop daily for a week, then increasing by one drop every week. Acetarsone in dose of 0 25 gram 2 to 3 times daily for a week may be given. Suramin (Germanin) or Bayer 205 may be used with success.

For temporary relief 10 p c calcium gluconate may be injected intravenously.

In herpes gestationis is advocated autohæmotherapy or injection of 10 c c of inactivated auto serum. Progesterone injection has also been advocated in this condition. Combined cortisone and progesterone therapy is also advocated, Proluton 50 mg daily for 7 days, then 30 mg, 25 mg, 15 mg daily is sometimes valuable in herpes gestationis. Cortisone (Roussell) 100 mg is given daily for 3 days, 75 mg for 6 days and 50 mg cortisone is continued with 100 mg progesterone daily. Autohæmo therapy with 10 c c of blood has also been advocated. Subcutaneous injection of blood serum from healthy pregnant woman gives good results.

Sulphapyridine (May & Baker) is given as ½ tablet every 6 hours for a week and then every 12 hours for a long time with regular checking of the blood picture. Antihistamines may also be used for a week or 10 days. Diazone or sulphoxone sodium (Abbott) in dose of one tablet every 8 hours for a week, later on twice daily for a week and is continued for a long time with one tablet every day keeping a check on the blood picture and hæmoglobin. Dapsone or diamino phenyl sulphone is the drug of choice. It is used in dose of 100 mg daily by mouth in divided doses.

Antibiotics—Penicillin crystalline 'G' may be given by injection when scratching causes secondary pyogenic infection and systemic reactions Aureomycin may be used orally in dose of one capsule every 6 hours for 4 days together with vitamin B complex

Locally warm Condy's bath is helpful In severe itching Liniment Calamine with 1 per cent phenol is used Ointment may be used such as

(1)	Sulphur ppt	gr 20
	Zinc oxide	dr ½
	Vaseline Alba	oz 1
	Ung for external use	
(2)	Hydrarg Ammon	gr 20
	Zinc oxide	dr ½
	Vaseline Alba	oz 1
	Ung for external use	

(3) 3 p c Aureomycin ointment as well as other antibiotic ointments have sometimes been used

(4) Hydrocortisone (Roussell) ointment locally allays itching and produces soothing effect

Diet—Lacto vegetarian diet has proved of value in tropics Those who are non vegetarian it is advisable for them to avoid egg lobster and crab in food

PEMPHIGUS

Definition Is a chronic and relapsing skin disease associated with bulla formation on the normal skin and ending fatally sooner or later

Etiology Classification of pemphigus —

(1) Pemphigus acutus
(2) Pemphigus vulgaris inocuous
(3) Pemphigus vulgaris chronicus
(4) Pemphigus vegetans
(5) Pemphigus erythematode
(6) Pemphigus foliaceus
(7) Pemphigus neonatorum
(8) Ocular pemphigus
(9) Familial benign chronic pemphigus

Profession may be responsible for the acute type of the disease as it has been observed that those who handle carcaces and butchers suffer from it Sometimes toxæmia is held responsible for pemphigus acutus Pemphigus neonatorum is due to staphylococal infection of the skin in infants and is now called Impetigo neonatorum Familial benign chronic pemphigus is supposed to be a form of Darier's disease and is associated with vitamin A deficiency Cause is not known for other types of pemphigus Some believe that the disease is a metabolic disorder Virus is also held responsible for the causation of pemphigus

Age—Acute pemphigus is common in young adults Pemphigus neonatorum is found in new born children Other cases of pemphigus are generally found in elderly people past middle life but in the tropics it affects round about the 40th year of age Sex—affects both sexes but in the tropics seems to be common amongst males

Signs and symptoms **Pemphigus acutus**—It is a rare skin disease Onset is sudden There is always

present a history of having had a trauma during handling of dead bodies or having had vaccination Bullæ first appear on the neck and inside the mouth and spread rapidly all over the body (Fig No 66) in successive



Fig No 66
Pemphigus acutus

crops Bullæ may be round like half hen's egg in size It may be tense or flaccid Sometimes bullæ coalesce together Contents of the bulla is *serous to* start with but becomes purulent later on Sometimes the bulla content may be hæmorrhagic also The bulla formation becomes generalised and covers the whole body including the mucous membranes of mouth, nostrils eyes, rectum and in woman vagina Bulla ruptures soon leaving a raw and oozing surface

Temperature is very high even from the beginning Patient generally becomes stuporose and dies within the first week of the attack Rarely the first attack subsides only to relapse after a month and the patient succumbs to it

Diagnosis (1) History of injury in a butcher or having had vaccination (2) High temperature,

(3) Bulla formation, (4) Cytology of the bulla floor shows no acantholysis, (5) Culture of the bulla fluid shows no organism by Gram's stain and most of the cells are polymorph with very few eosinophil cells if found at all, (6) Culture of bulla fluid is sterile, (7) Biopsy—histopathology of acute pemphigus is characterized by the formation of a bulla at the dermo epidermal junction or in the middle of the stratum mucosum Tzanck cells are also found which are characterized by the persence of rete cells in the vesicle

Differential Diagnosis (1) *Dermatitis herpetiformis*, (2) Erythema multiform bullosum, (3) Drug rash

Prognosis Is very grave Always ends fatally

Treatment *Prophylactic*—to avoid injury in butchers and to dress surgically when there is any trauma in such a profession

Curative—Aureomycin (250 mg) capsule is given every 6 hours with vitamin B Complex as a routine measure Corticotropin (ACTH) aquous solution is administrad by I M injections in dose of 40 units every 4 hours for the first 2 days and then every 6 hours *Blood transfusion* is also given and may be repeated daily if needed Cortisone may be given (25 mg) tablet every *4 hours for 5 days and then every* 6 hours Continuous warm water bath with dilute *Condy's lotion* is very relieving

Diet—Liquid diet is given with high protein such as eggs meat extractives, liver diet Salt should not be given during ACTH or cortisone therapy K salt (Calcutta Chemical) may be used instead of ordinary table salt

(2) **Pemphigus vulgaris inocuous**—Is the benign type of pemphigus Patients are often admitted as cases of dermatitis herpetiformis but gradually develops the clinically developed type of pemphigus vulgaris chronicus This type of pemphigus is commonly seen in the tropics Age—no age is exempt Sex—found in both sexes

Signs and symptoms few bullæ may develop on the normal skin (Fig No 67) whithout any areola The



Fig No 67
Pemphigus vulgaris inocuous
(Case of Dr K C Kandhari)

lesions are half pea to half grape in size Lesions are often itchy Nikolosky s sign is present which is believed to be due to a duced calcium content of the epidermis It is a recurrent condition Leaves no scar or pigmentation on healing

Diagnosis (1) Recurrent type of bullous lesion which are itchy but leaves no scar or pigmentation on healing (2) Nikolosky s sign is positive (3) Biopsy—histopathology shows a subepidermal bulla (Fig No 68) at the beginning of the disease but later the bulla become intraepidermal (Fig No 69) with acantholysis (4) Cytology shows acantholytic cells There may be round cells, most of which are poly but few may be eosino The number of eosinophil cells do not increase on

subsequent examinations, (5) Bulla fluid smear examination with Gram stain shows no bacteria, (6) Bulla fluid culture is sterile, (7) Blood Wassermann and Kahn tests are negative, (8) Diminished output of urinary 17 Ketosteroid is considered to be an early diagnostic point

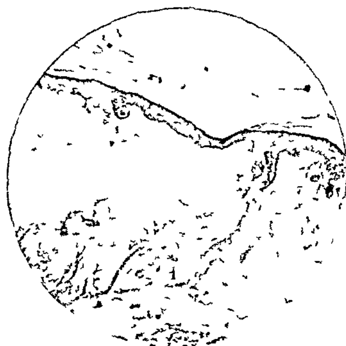


Fig No 68

Histopathology of Pemphigus vulgaris mocuous showing subepidermal bulla

(Case of Dr B N Banerji & Dr K D Lahiri)

Prognosis Fair but after several years develops into pemphigus vulgaris chronicus and the patient dies

Treatment Prophylaxis—nothing is known

Curative—Sulphapyridine tablet is given as one tablet 4 hourly for a week Aureomycin (250 mg) capsule is given with Vitamin B complex every 6

hours for a week Autohæmotherapy—starting with 5 c c of patient s own blood on alternate days increasing by 0 5 c c every time until 10 c c is given

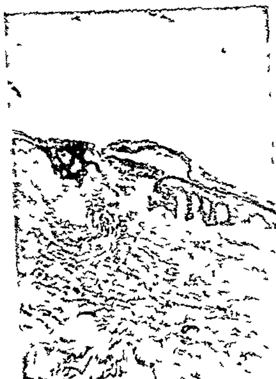


Fig No 69

Histopathology of Pemphigus vulgaris ...
showing subepidermal bulla later becoming intra pidermal
(Case of Dr B N Banerji & Dr K D Lahiri)

Vitamin C (500 mg) is given by injection twice daily and Vitamins A and B Complex are given by mouth Cortisone (25 mg) tablet is given by mouth every 6 hours for 2 days then reduced to 10 mg every

6 hours and ultimately 5 mg 6 hourly, 8 hourly, 12 hourly and once daily before it is stopped Corticotropin (ACTH) is given in the gel form in dose of 20 units twice daily for seven days and then gradually reduced to 10 units twice daily, 5 units twice daily and then once daily before it is finally stopped One or two blood transfusions are helpful

(3) **Pemphigus vulgaris chronicus**—may occur as it is or may develop from a case of Pemphigus innocuous Age—may occur at any age Sex—found in both sexes Quite common in the tropics

No cause is known

Signs and symptom Sudden development of one or many bullæ on the normal skin There is no areola round the bulla In size it may be half pea to half grape or hen's egg Bulla may be tense or flaccid (Fig No 70) Nikolosky's sign is positive *Bulla fluid* is serous to start with but becomes purulent soon Sometimes bulla fluid is also hæmorrhagic in nature Situation of the bullæ are in the axilla, groin and may be anywhere on the body and mucous membrane Sometimes the bulla on the palate and tongue may appear first Bullæ may be found in the nostrils and in the vagina and rectum causing prolapse of the uterus and prolapse of rectum also When the bulla ruptures a raw oozing surface is left which is very tender and crust may form Patient may develop fever late in the disease and loose weight

Diagnosis (1) Sudden appearance of bulla with positive Nikolosky's sign, (2) Loss of weight and the

peculiar mousey smell of the patient, (3) Biopsy—histopathology shows an intraepidermal bulla with acantholysis



Fig No 70
Pemphigus vulgaris
(Case of Dr B N Banerji & Dr K P Lahiri)

(Fig No 71) (4) Cytology of the bulla floor shows acantholysis of cells There are round cells which are polymorphonuclear in type but few eosinophil cells may be found, (5) Bulla fluid shows the presence of no organism with Gram s stain (6) Bulla fluid in culture is sterile, (7) Serum protein estimation shows low albumin and high globulin Total protein is low (8) Blood Wassermann and Kahn tests are negative (9) Diminished urinary output of 17 Ketosteroid occurs quite early in disease

Differential Diagnosis (1) Dermatitis herpetiformis, (2) Drug rash, (3) Secondary syphilis



Fig No 71

Histopathology of Pemphigus vulgaris showing bulla in the epidermis and round cell infiltration in the dermis

(Case of Dr B N Banerji & Dr K D Lahiri)

Prognosis Is grave Patient dies within 2 years Rarely survives 8 to 10 years with treatment

Treatment Prophylactic—nothing is known

Curative—Suramin is injected intravenously on alternate days in dose of 0 2, 0 3, 0 4, 0 5, 0 6, 0 7, 0 8, 0 9, 1 0 g, 1 0 g

Acetarsol orally every morning for 3 days, rest for 3 days and repeating for 3 days, in dose of 0 25 g tablet

Aureomycin (250 mg) capsule is given every 6 hours with Vitamin B complex for a week or so or Chloramphenical 0 5 g 4 times daily with Vitamin B complex for 2 weeks Cortisone tablet (25 mg) by mouth every 6 hours with Vitamin B complex orally for a week is helpful and then gradually the dose is reduced ACTH may be given in dose of 40 units of aquous solution every 6 hours for a week and then 20 units ACTH gel may be given twice daily for 4 days and then gradually reducing until stopped Bayer 205 by intravenous injection biweekly or Suramin may be given a trial Repeated blood transfusion (250 c c) weekly or biweekly is helpful

Pemphigus vegetans—Etiology Is very rare but they are seen in the tropics Both sexes suffer but is common in women

Signs and symptoms Malaise and often slight fever preceeds the formation of bullæ The onset is insiduous in pemphigus vegetans Bullæ are generally seen inside the mouth lips and nose first but may appear on the body before they come out inside the mouth The lesions on the body are seen around the genitalia, groin perineum exællа, extremities and on the scalp The bulla ruptures and the base of the bulla becomes papular and gets covered by a crust Sometimes groups of blebs are seen which rupture and the bases proliferate to form vegetating masses (Fig No 72) when it is clinically a case of pemphigus vegetans Nikolosky's sign is present Gradually the patient looses weight Death is due to some intercurrent disease

Diagnosis (1) Formation of bullæ in the inguinal, perineal and genital regions, (2) Vegetating type of

lesions, (3) Biopsy—histopathology shows imperfect keratinisation of the stratum corneum, acanthosis and



Fig No 72
Pemphigus Vegetans
(Case of Dr B N Banerji & Dr K D Lahiri)

edema in the stratum mucosum Bulla is intraepidermal but may also be subepidermal Papillae are hyper trophied and micro abscesses are found in the papillary bodies with eosinophil cells There is a compact mass of infiltration in the dermis with eosinophil cells which is very characteristic (Fig No 73), (4) Cytology shows presence of eosinophil cells with acantholysis Tzanck cells also are found, (5) Bulla fluid smear examination after staining with Gram s stain will not show the presence of any bacteria, (6) Bulla fluid culture is sterile, (7) Blood protein—total protein is diminished very much Albumin

is decreased and globulin is increased, (8) Blood chemistry—sugar chloride and calcium are lowered, (9) Blood Wassermann and blood Kahn tests are negative, (10) Diminished urinary output of 17 Ketosteroid is an early diagnostic finding



Fig No 73

Histopathology of Pemphigus vegetans showing intraepidermal bulla with thickening of the prickle cell layer and micro abscesses

(Case of Dr B N Banerji & Dr K D Lahiri)

Differential diagnosis (1) Condylomata lata, (2) Condylomata accuminata (3) Bromide and iodide (fungating) rash (4) Dermatitis vegetans, (5) Other granulomas of skin

Pragonsis Is fair so far as the longivity is concerned compared with other types but the patient succumbs to some undercurrent disease May change to P vulgarls

Treatment Prophylactic—nothing is known

Curative—Arsenic therapy is used Carbarson 0 25 g tablet thrice daily may be given Sulpha drug such as sulphapyridine or sulphadiazine may ba given by mouth as one tablet 6 hourly for a week Antibiotic such as crystalline penicillin G' in aquous solution is injected in dose of 5 lacs daily for a week or so Aureomycin capsule (250 mg) every 6 hours together with Vitamin B complex is advocated for a week or ten days and then repeated after an interval of a week

Locally when there are bullous eruptions present Liniment Calamine with 1 p c Hydrarg Ammon may be applied every hour When the bullae rupture 1 p c Gentian violet is painted followed by 1 p c Ung Hydrarg Ammon dressing may be given Antibiotic ointment may be helpful

Injection of 2 c c Crude liver extract is given daily Repeated blood transfusions (250 c c) biweekly is very helpful

(5) **Pemphigus erythematode** *this is also known* as Snear Usher Syndrome

May occur at any age but is commonly found in young adult males in the tropics

Signs and symptoms Erythemato squamous patches may occur at intervals on the scalp, face or trunk in the intrascapular region and on sternum These bullae rupture and a raw, eczematous area resembling lupus erythematosus or seborrhoic eczema may be seen Sometimes minute vesicles are seen at the periphery

of the these eczematous lesions (Fig No 74) May develop into pemphigus vulgaris



Fig No 74

Pemphigus erythematode

(Case of Dr B N Banerji & Dr K D Lahiri)

Diagnosis (1) Erythemato squamous or vesiculo squamous lesions on the scalp face intra scapular region and sternal region (2) Follicular plugging ? the lesion (3) Nikolosky s sign i positive, (4) Biopsy — histopathology shows acantholysis of cells in the stratum mucosum Dyskeratotic cells are found in the stratum granulosum There is no follicular hyperkeratosis Liquefactive degeneration of the dermoepidermal junction is characteristic (Fig No 75) (5) Diminished urinary output of 17 Ketosteroid is an early finding

Prognosis is best amongst its types and life may be as long as 10 or 12 years Prognosis is grave

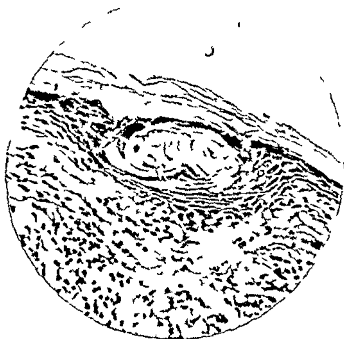


Fig No 75

Histopathology of Pemphigus erythematode showing bulla in the epidermis and round cell infiltration of the dermis and flattening of the papillae

(Case of Dr B N Banerji & Dr K D Lahiri)

when the lesions start on the mucous membrane of the mouth or change into P vulgaris

Differential Diagnosis Lupus erythematosus, (2) Seborrhoic dermatitis, (3) Erythema multiforme

Treatement Prophylaxsis is not known Crude Liver extract injection and Vitamin A and C are helpful

Curative—*Antibiotics* can be used *orally* and locally Blood transfusion is indicated when condition becomes bad

(6) **Pemphigus Foliaceous** Etiology It is a rare disease Is also seen in the tropics Affects adults Cause is not known

Signs and symptoms Bullæ appear which are flaccid in type Bulla spreads by peripheral extension Nikolosky s sign is positive Bullæ develop symmetrically and cover the whole body, extramities and face and even the mucous membranes of the eyes and mouth are involved Bullæ rupture and raw area is left Over the raw area flake like lesions develop The patient presents an exfoliative condition of the skin (Fig No 76) At intervals pemphigus foliaceous may change into pemphigus vulgaris chronicus



Fig No 76

Pemphigus Foliaceous

(Case of Dr B N Banerji & Dr K D Lahiri)

Diagnosis (1) Exfoliative condition of the whole body, (2) Nikolosky's sign positive, (3) Biopsy—histopathology shows intraepidermal bulla formation which may be subcorneal Acantholysis is commonly seen Imperfect keratinization of the cells of the stratum corneum is seen showing parakeratosis Acanthosis of stratum mucosum There is edema in the dermis with round cell infiltration which are of lymphocytic, neutrophilic and histiocytic in types, (4) Blood picture shows eosinophilia, (5) Blood W R and Kahn tests are negative, (6) Blood protein—albumin is decreased and globulin is increased, (7) Diminished urinary output of 17-Ketosteroid is an early sign of value

Prognosis is same as pemphigus vegetans

Treatment same as pemphigus vulgaris chronicus

Locally Hydrarg Ammon ointment may be helpful Blood transfusion is the sheet anchor of treatment Other treatments are supportive

(7) **Pemphigus neonatorum**—Is impetigo contagiosum of the new born (see impetigo)

(8) ***Ocular pemphigus***—*Is said to be a* clinical entity and is dealt in books of ophthalmology In the tropics almost every case of pemphigus develops ocular lesions during the course of the disease (Fig No 77) but clears away with the improvement of the skin condition

Prognosis Does not become blind if properly treated

Treatment 2 5 p c cortisone eye drop and eye ointment are valuable Blood transfusion for the general condition helps to clear the eye lesions also

(9) Familial benign chronic pemphigus is also known as Haily Haily disease—Is seen in several



Fig No 77
Pemphigus of the eyes
(Case of Dr B N Banerji & Dr K D Lahiri)

members of a family Deficiency of vitamin A is supposed to be the etiological factor Males are commonly affected

Signs and symptoms Recurrent bullous lesions develop on the sides of the neck and intertrigenous areas with positive Nikolosky's sign Mucous membrane lesions are not found Patient s health is not impaired

Diagnosis (1) Recurrent localised bullous rash on several members of a family, (2) Nikolosky s sign is positive, (3) Biopsy – histopathology shows dyskeratosis

of cells and partial acantholysis Bulla occurs lower down in the stratum mucosum

Prognosis Is good

Treatment Vitamin A in high doses when given for a long time keeps the disease under control Locally 1 p c Hydrarg Ammon *ointment is all that is necessary*

Pemphigoid—The bullous rash of erythema multiforme bullosum and dermatitis herpetiformis are sometimes called 'pemphigoid' from the benign nature of these rashes

EPIDEMIC DROPSY

Definition Is a tropical disease characterized by sarcoid like lesions and with pigmentation and erythema over *the skin accompanied with edema of legs* and some times with fever and palpitation

Etiology This is due to cooking with mustard oil contaminated with argemone oil The disease occurs in epidemic form at different places in the tropics Age—people *of all ages are affected,* Sex—both sexes are affected

Signs and symptoms Starts as a gastro intestinal disturbance followed by fever Pallor appears together with engorged neck veins and edema of legs Gradually multiple telangiectasia like spider nævi appear all over the body The skin shows alternate bands of erythema and blanching on pressure with spread out fingers Erythematous gyrate patches are seen (Fig No 78) Hyperpigmentation of the face and extremities are commonly seen Pin head to pea sized nodules and

sarcoid like lesions appear on the scalp, face and body



Fig No 78
Epidermic dropsy
Showing erythematopapular lesions on the arm and forearm

(Fig No 79) which rarely ulcerates Papillomatous growth is found on the mucous membranes and on tongue



Fig No 79
Epedermic dropsy
nodules on back
(Case of
Dr L. K. Ganguli)

(Fig No 80) and at other places Breathlessness and palpitation are usual accompainment

Diagnosis (1) Typical pigmentation, erythema, blanching and redness on pressure with sarcoid like



Fig No 80
Epidemic dropsy nodule on mucous membrane of lip
(Case of Dr L K Ganguli)

nodules or spider naevus like lesions, (2) History of consuming mustard oil, (3) Examination of mustard oil for the presence of argemone oil, (4) *Histopathology* shows only dilatation of vessels in the dermis

Prognosis Good

Treatment Prophylaxis is to stop consumption of mustard oil Curative is to stop mustard oil and to give the patient high protein diet and vitamins by mouth Liver extract 2 c c I M injection is given daily Ephedra vulgaris dr ½ is given in water thrice daily after food Intravenous inj of 25 p c glucose with 10 p c Calcium and Vitamin C (500 mg) may be given every day Rest in bed in essential No local treatment is necessary for the skin lesions

CHAPTFR VIII

BACTERIAL SKIN DISEASES

Skin diseases may be caused by different bacteria producing different types of lesions

The organisms causing disease like

1 Pyogenic infection
2 Tropical ulcer
3 Chancroid
4 Cutaneous anthrax
5 Cutaneous diphtheria
6 Rhinoscleroma
7 Leprosy
8 Cutaneous tuberculosis

Pyogenic Infection This forms a group of skin infectious caused by strepto and staphylococci Common pyogenic infections in the tropical country are

(a) Impetigo contagiosa
(b) Pemphigus Neonatorum
(c) Bochart s impetigo
(d(Furunculosis
(e) Carbuncle
(f) Chronic folliculitis
(g) Summer boils
(h) Dermatitis vegetans
(i) Acrodermatitis perstans and Acrodermatitis tropicalis
(j) Granuloma pyogenicum

Impetigo Contagiosa Definition—Is an acute contagious superficial, bacterial, skin disease characterized by the formation of vesicles and crust formation

Etiology Caused by hæmolytic group A strepto cocci and staphylococci as a secondary invader but sometimes by both the organisms Impetigo on the scalp *is due to scratching in* pediculosis and on the body is due to scratching in scabies Age—No age is exempt Sex—common in both sexes Acrodermatitis enteropathica and pemphigus neonatorum affects infants whereas impetigo and granuloma pyogenicum affects the children and all others affect adults in the tropics

Signs and symptoms They cause both systemic reaction and damage to the skin Acute impetigo contagiosa often appears on the face of children (Fig No 81) and the infection is due to contact A vesicle appears with an areola and it bursts and the serum gets dried up and remains on the denuded surface of the skin as a stuck on crust (Fig No 82) Several bullae appear together and is known as **Impetigo bullosum** (Fig No 83) There may be systemic reaction also Bullæ may be come generalised also The generalisation of the impetigo takes place in the new born infants with systemic reactions when it is called **Impetigo neonatorum** or Pemphigus neonatorum The bulla in an impetigo ruptures and at the periphery of the denunded surface a group of bullae appear causing extension peripherally This type is called **Impetigo sercinatum** (Fig No 84) When the impetigo extends deep down to the dermis it is called **Ecthyma** **Ethyma Contagiosum** (Orf) is a virus disease of sheep and goat and is occasionally transmitted to man When the *impetigo* extends

both peripherally and deeply it is called **Impetigo ulcerativum** Impetigo may produce a circular or



Fig No 81
Impetigo contagiosa

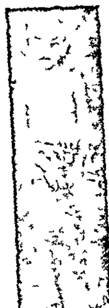


Fig No 82
Impetigo contagiosa
(Lesions are on face, hands and feet)

oval erythematous lesions on the face of a child and become chronic when it is called **Impetigo pety roides** When the impetigenous lesion involves the superficial part of the hair follicle it is called **Bochart's impetigo** (Fig No 80) Affects also the hairy regions involving the whole of the hair follicles when it is called **Folliculitis** May involve any hairy region When chronic and affects beard region it is called **Sycosis barbæ** When affects the nuchal region it is known by the name of **Sycosis nuchæ** and there may

develop keloid which is called **Nuchal keloid** (Fig No 86) When the lesion involves the whole of the hair

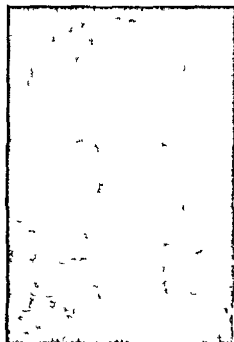


Fig No 83
Impetigo bullosum

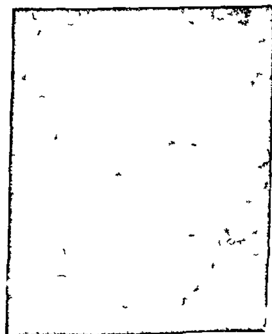


Fig No 84
Impetigo sercinatum

follicle and produces gangrene of the hypodermis it is called **Furunculosis** which occurs most commonly on the

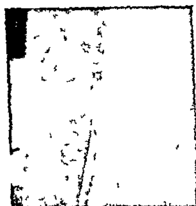


Fig No 85
Bochart's impetigo

neck When several of the hair follicles are involved producing gangrene of a large portion of the hypoderm resulting in systemic reaction it is called **Carbuncle** Generally affects people in the tropics at 40 years of age and having high blood sugar This condition is associated with grave systemic reaction When suppurates looks

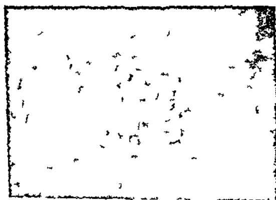


Fig No 86
Nuchal keloid
(Back of head)

like a honeycomb The infection goes down to the hair follicle and apocrine glands The condition clinically presents a picture of skin coloured nodules Which

suppurates and is known as **Hidradenitis suppurativa** Axillæ are the common sites There may be systemic reaction with it When the infection becomes chronic it affects different apocrine glands and affects the sweat glands including the subcutaneous tissue and fascia When the infection goes down the spiral ducts and affects the sweat glands the picture is that of multiple pea sized to half beetle nut sized skin coloured lesions which affects mainly the face, neck, shoulders and even the chest and rarely the arms It is a very painful condition The lesions change colour from that of the skin to reddish or reddish brown and suppurate Commonly affects children in the tropics but sometimes seen in adults also in summer This condition is called **Summer Boil** or *Mangoe boil*

Rarer types of pyogenic lesions are the following —

The organism produces a low grade localised chronic infection particularly in the intertrigenous areas such as the crural regions, the axillae or even on the skin of the extremities The lesion looks granulomatous, warty or papilomatous with pus oozing out and producing a cauliflower like growth when it is called **Dermatitis vegetans** Patients generally suffer from malnutrition and with history of the skin lesion of over 3 to 6 years duration ***Dermatitis gangrenosum*** *is the gangrene of* the skin and occurs in association with small pox, typhoid, carbuncle,erysepelas, gas gangrene, in vascular diseases like Buerger's disease, Raynourd's disease arteriosclerosis as in diabetis and also in embolism and thrombosis of arteries After a trauma in the periony chial region a low grade bacterial infection causes a very slow forming, superficial, undermined skin lesion which proceeds generally from the thumb or the toe over the palm or sole and is

resistant to all treatment This condition is called **Acrodermatitis perstans** (Hallopeau) or Dermatitis repens (Crocker) which is generally unilateral But in the tropics a similar chronic slowly active, symmetrical lesion either on both palms or both soles occur which may be called **Acrodermatitis tropicalis** In the tropics repeated gastro intestinal infection in children causes stunted growth and even the development of a symmetrical chronic pyogenic moist lesions which travel down from the genital clefts to either side of the medial sides of the thighs involving both the gluteal regions and reach down the knees ankles and the dorsum of feet Together with this there may be alopecia and nail changes with thrush like lesion on the tongue and buccal mucosa may be present The skin lesion may be present at other places e g circumoral region eye lids and also on the upper extremities This condition is seen in undernourished children in the tropics with a history of commencement of the disease at infancy and is called **Acrodermatitis enteropathica** (Fig No 87) A type of rapidly growing pedunculated growth the size of a pea or a beetle nut may occur anywhere on the skin after a microscopic trauma This is due to the entrance of the pyogenic organisms causing a flesh coloured growth This is moist with discharge Injury causes bleeding from its rough surface May occur on face back hands and feet This condition is known as **Granuloma Pyogenicum** (Fig Nos 88 & 89) Sometimes multiple nodular chronic painful lesions with necrosis at the centre occur on the face and is called **Staphyloderma chronica** or **Staphyloma** (Fig Nos 90 & 91)

Diagnosis (1) Clinical examination, (2) Smear

suppurates and is known as **Hidradenitis suppurativa** Axillæ are the common sites There may be systemic reaction with it When the infection becomes chronic it affects different apocrine glands and affects the sweat glands including the subcutaneous tissue and fascia When the infection goes down the spiral ducts and affects the sweat glands the picture is that of multiple pea sized to half beetle-nut sized skin coloured lesions which affects mainly the face, neck, shoulders and even the chest and rarely the arms It is a very painful condition The lesions change colour from that of the skin to reddish or reddish brown and suppurate Commonly affects children in the tropics but sometimes seen in adults also in summer *This condition is called* **Summer Boil** or *Mangoe boil*

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The organism produces a low grade localised chronic infection particularly in the intertrigenous areas such as the crural regions, the axillae or even on the skin of the extremities The lesion looks granulomatous, warty or papilomatous with pus oozing out and producing a cauliflower like growth when it is called **Dermatitis vegetans** Patients generally suffer from malnutrition and with history of the skin lesion of over 3 to 6 years duration ***Dermatitis gangrenosum*** *is the gangrene of* the skin and occurs in association with small pox, typhoid, carbuncle,erysepelas, gas gangrene, in vascular diseases like Buerger's disease, Raynourd's disease arteriosclerosis as in diabetis and also in embolism and thrombosis of arteries After a trauma in the perionychial region a low grade bacterial infection causes a very slow forming, super *ficial,* undermined skin lesion which proceeds generally from the thumb or the toe over the palm or sole and is

the presence of inflammatory sign in the dermis and hypoderm is seen in summer boil In dermatitis vegetans

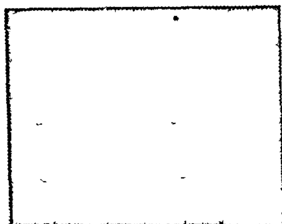


Fig No 89
Granuloma pyogenicum
(Nodule on the back of axilla)



Fig No 90
Staphyloma

the important changes in the epidermis are micro abscesses and pseudohyperplasia In acrodermatitis

examination with Gram's stain will reveal strepto and staphylococci, (3) Culture of the discharge will show the

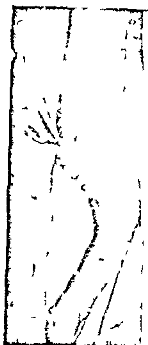


Fig No 87
Acrodermatitis enteropathica

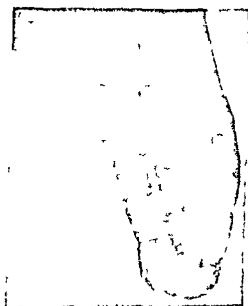


Fig No 88
Granuloma pyogenicum
(Nodular growth on the tip of thumb)

pyogenic organisms (4) Biopsy histopathology will show in impetigo contagiosa an intra epidermal bulla high up in the stratum corneum The lesion reaches up to the dermis The lesion is in the upper part of the hair follicle in *Bochart's impetigo* *Inflammation* of the whole of the hair follicle is found in folliculitis whereas in furunculosis there is inflammation of the hair follicle together with gangrene of the hypoderm When there is inflammation in more than one hair follicle together with gangrenous change of the greater part of the hypoderm it is the picture of carbuncle There is *dilatation* of the sweat gland together with

pemphigus vegetans drug rash (bromoderma) and tuberculosis verucosum cutis (4) Acrodermatitis tropicalis of palm and sole has to be differentiated from psoriasis palm and sole, (5) Acrode matitis entero pathica from psoriasis, (6) Granuloma pyogenicum from haemangioma and subungual melanoma

Prognosis Is good in every case when properly diagnosed and systematically treated

Is good in granuloma pyogenicum with excision

Treatment Prophylaxis—In the tropics prophy laxis is very difficult With the raising of the standard of living and better knowledge of scientific diet and hygiene various skin diseases which are due to infection and trauma are not likely to occur so much

Curative—Impetigo is treated by removing the bullae roof under aseptic condition followed by washing with boiled warm water and a bland soap Then it is dressed every hour with the lotio No 1

No 1	Argentii Nitras	gr 5
	Aqua Distil	oz 1

Lotio for external use Supply oz20 in a coloured phial Linen soaked in this lotion is applied every hour for a day or two when the lesions get dried up When dry lotion No 2 is painted once daily

No 2	Gentain violet	gr 5
	Aqua Distil	oz 1
	Lotio for external use	

tropicalis there is acanthosis of the stratum mucosum with papillomatosis and there may be found micro



Fig No 91
Histopathology of Staphyloma

abscesses Granuloma pyogenicum shows dilatation and proliferation of vessels in the dermis together with infiltration with polymorphonuclear cells and mast cells

Differential Diagnosis (1) Impetigo is to be differentiated from herpes zoster, chicken pox urticaria bullosum, dermatitis herpetiformis and epidermolysis bullosum, (2) Impetigo peteroides has to be differentiated from seborrhoic dermatitis and contact dermatitis, (3) Dermatitis vegetans has to be distinguished from

pemphigus vegetans, drug rash (bromoderma) and tuberculosis verucosum cutis, (4) Acrodermatitis tropicalis of palm and sole has to be differentiated from psoriasis palm and sole, (5) Acrodermatitis entero pathica from psoriasis, (6) Granuloma pyogenicum from haemangioma and subungual melanoma

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	Aqua Distil	oz 1
	Lotio for external use	

When it gets dried up the skin is kept bandaged up with an ointment No 3 or Zichthol (Gantosan)

No 3	Ichthyol	gr 5
	Vaseline alba	oz 1

Ft Ung for external use At night dressed only with ointment No 4

No 4	Hydrarg Ammon	gr 10
	Pulv amylum	dr 1
	Zinc oxide	dr 1
	Vaseline alba	oz 1

Ft Ung for external use

No 5	Ichthyol	dr 4
	Glycerine pure	dr 4
	Ft paint for external use	

When there is systemic manifestation of toxicity like fever sulphadiazine or Elkosin is given for 4 days orally 0 5 g at 6 hours for adult but rarely Penicillin crystallin 'G' is injected in 0 2 Mega Unit dose twice daily for 5 days only In impetigo peteroides only ointment No 4 is applied In folliculitis in the acute stage hot Condy's bath followed by shaving of the part is essential and then painting twice daily of the lotion (No 2) followed by application of an ointment (No 3) and another ointment (No 4) only at night When chronic crude liver extract is injected biweekly in dose of 2 c c I M Carbuncle is treated by hot fomentation 4 times daily with injection intramuscularly twice daily of 0 2 Mega unit of Crystalline penicillin 'G' for 5 to 7 days When sloughing occurs S S Magsulph dressing is helpful When slough clears

away dressing is done with an ointment (No 4) Hydradinitis suppurativa is treated with hot fomentation followed by application of a paint (No 5) locally applied 4 to 6 times daily together with Sulpha therapy or Penicillin injections Sometimes requires excision of the glands Summer boils should be treated by hot fomentation and application of a paint (No 5) together with sulpha drug Rarely needs operation

Dermatitis vegetans should be treated internally by high vitamin C and locally with Condy's bath and a lotion (No 2) is painted followed by the application of an ointment (No 3) and after 2 to 4 day treated with another ointment application (No 4) Antibiotic may be used orally Acrodermatitis tropicalis requires locally an ointment (No 6)

No 6	Acid salicylic	gr 10
	Zinc oxide	dr 4
	Liq Picis carb det	m 10
	Vaseline alba	oz 1
	Ung for external use	

Later on painting the part daily with a paint (No 7) and dusting over with a powder (No 8) is helpful

No 7 10 p c Crude Coal tar in acetone for external use

No 8	Pulv zinc oxide	dr 3
	Pulv acid salicylic	dr 1
	Pulv Amylum	ad oz 1

In acrodermatitis enteropathica orally is given diodoquin (diiodohydroxyquinoline) 100 mg four times daily

for a week, 100 mg thrice daily for a week 100 mg twice daily for a week and ultimately 100 mg once daily for another week before stopping the drug Locally treated with painting of a lotion (No 2) followed by application of an ointment (No 3) during the day and only the application of another ointment (No 4) is advocated at night High protein diet and multivitamin (Panlyn of Calcutta Chemical) are advocated

In granuloma pyogenicum removal of the growth by excision followed by the application at the base of Tr Iodine or Silver nitrate stick is essential X ray therapy is sometimes advocated

TROPICAL ULCER

Definition Is a chronic ulcer of the leg occuring in tropics due to fusiform bacillus and spirocheta of Vincent

Etiology Commonly seen during the monsoon all over the tropics Age—any age Sex - Common in males Trauma is a predisposing factor but malnutrition particularly of protein and vitamin C is the main etio logical basis in the causation of tropical ulcer Various organisms are found such as staphylococci streptococci but the predominating organisms are fusiform bacillus and spirocheta vincenti

Signs and symptoms After some trauma starts as a red papule on which a bleb of the size of a pea is formed The bulla ruptures leaving a small ulcer The ulcer is round with an undermined irregular edge The base of the ulcer may be covered by a crust or a pseudo membrane The ulcer is usually a 1 ole

but multiple ulcers may be seen Site is commonly the leg and other exposed parts like the dorsum of the hands Ulcers may be superficial but sometimes it may extend deeply destroying the underlying structures like muscles, tendons and even affects the periosteum and the bone It is a tender and painful sore There may be systemic reaction with it

Diagnosis (1) Indolent solitary ulcer on the leg in a tropical country (2) Ulcer is tender and is covered with a pseudo membrane and there is a red hallow, (3) Smear examination from the ulcer will show fusiform bacilli and spirocheta vincenti (4) Vitamin C in blood is subnormal (5) Serum protein shows low total protein with low albumin, (6) Biopsy—histo pathology shows edema in the dermis with many fusiform bacilli

Differential diagnosis (1) Diphtheretic ulcer, (2) Varicose eczema, (3) Syphilitic ulcer (4) Scabies

Prognosis Good with rest and proper treatment,

Treatment Prophylactic—patient should avoid trauma Regular use of foot wears is helpful Nutrition must be improved Vitamins particular A and C should be taken in prophylactic dose

Curation—(1) Locally warm Condy s soaks 4 times daily followed by the application of Mag sulph pastes for day or two then an ointment (No 1) containing

No 1	Acreflavin	gr x
	Oil Morrhœ	oz 1

Antibiotics may also be used such as aureomycin one capsule (250 mg.) every 6 hours with Vitamin B complex orally or combiotic may be injected once daily 7 to 10 days. Same treatment is given to the bulbo when develops. Ducrey bacillus vaccine may also be injected.

CUTANEOUS ANTHRAX

Definition: is an acute specific skin disease characterized by a pustule on a wide erythematous base accompanied with systemic reaction and is caused by Bacillus anthracis.

Etiology: Anthrax although is rare these days but once or twice a year a patient is seen in a large hospital. Anthrax is not infrequently seen in the industrial areas dealing with raw hide and fur. Commonly seen in those who handle hide and fur. Cases of anthrax have occasionally been reported by using infected shaving brushes.

Signs and symptoms: Earliest lesion is a red elevated area of about ⅛ inch diameter. This red area becomes larger and a central black area appears called *eschar*. Surrounding this black area are tiny vesicles and this is surrounded by a red œdematous area and the whole lesion is called the '**Malignant Pustule**'. Together with the development of this lesion the part becomes tense and tender. Constitutional symptoms develop consisting of high temperature, headache, toxicity and albuminuria with prostration is the picture in acute cases. Lymphadenitis is present which often suppurates. Chronic cases commonly show enlargement of regional lymph glands. Rarely secondary lesions due to autoinoculation appear.

Diagnosis (1) Clinical picture (2) Smear from the vesicle or pustule shows the presence of B anthracis ; Which are rod shaped and squre shaped, gram positive bacillus, (3) Culture shows Bacillus anthracis (4) Blood examination for total count will show leucocytosis and high neutrophil count, (5) Histopathology—shows destruction of the epidermis and spongiosis Rarely an intraepidermal bulla is found There is oedema in the dermis and W B C and R B C are found present in it Anthrax bacilli are also seen

Differential Diagnosis (1) Impetigo, (2) Drug rash, (3) Erysipelas

Prognosis Is good with modern treatment

Treatment Prophylactic—sterilisation of shaving brush is essential In trade those who handle hide and fur should use gloves and aprons Prophylactic inoculation is valuable with Bacillus anthracis vaccine

In the management of a case of anthrax the case should immediately be hospitalized in special infectious disease ward with specially trained nursing staff

Patients should have rest in bed with plenty of fluid to drink and alkali to flush the kidneys Sulphadiazine or sulphapyridine in dose of 0 5 to 1 0 gm orally is given every 4 hours for a week with the examination of blood on every alternate days for agranulocytosis Together with this treatment Sclavo s serum 3 c c I V is given when available otherwise as a routine (1) Orally sulphapyridine 0 5 gram 4 hourly for 7 days (2) Combiotic injections intramuscularly for even days is helpful (3) Aureomycin (250 mg) capsule to be given

every 6 hours for two days then one every 6 hours for 5 days with vitamin B complex

Locally 1 p c Ung Ichthyol dressing is applied

CUTANEOUS DIPHTHERIA

Definition Is an ulcerated skin lesion caused by Cornybacterium diphtheriæ

Etiology All ages and both sexes may be affected Caused by C diphtheriæ Is found in 0 02 p c in the tropics

Signs and symptoms The lesions are commonly found on the inferior extremities The lesions may be eczematous or cellulitic or as chronic indolent ulcers There may be associated paralysis of the limb

Diagnosis (1) Indolent ulcers on legs with or with out paralysis (2) Biopsy—histopathology shows the inflammatory reaction with cornybacterium diphtheriæ, (3) Smear from the ulcer shows with special stain C diphtheriæ, (4) Culture of the pus from ulcerated growths for C diphtheriæ is positive

Prognosis Good if early diagnosed but paralysis may develop

Treatment Prophylaxis in suspected cases 20,000 i u antidiphtheretic serum is injected Curative 50,000 i u should be injected immediately and shou'd be repeated daily for 3 days in dose of 25,000 units Penicillin crystalline 'G' should be injected I M daily for 3 days in dose of 5 lacs

RHINOSCLEROMA

Etiology Rhinoscleroma is a chronic granulomatous skin disease of the nose caused by a gram negative organism called Bacteria rhinoscleromatosis

Etiology Is commonly found in central and upper India Seen commonly amongst young adults and found in both sexes Incidence is about 0 05 in the tropics

Signs and symptoms Starts as a red nodule on the side of the nose which grows very slowly and invades the nares and ulcerates (Fig No 92) The foul nasal discharge is the early symptom but later on obstruction develops due to growths in the nose nasopharynx and also in the larynx



Fig No 92
Rhinoscleroma

Diagnosis (1) Clinical features, (2) Biopsy—histopathology shows fairly large number of vacuolated histiocytes called 'Mikulicz cell' containing

every 6 hours for two days then one every 6 hours for 8 days with vitamin B complex

Locally 1 p c Ung Ichthyol dressing is applied

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LEPROSY

The general practitioner should consider leprosy like any other bacterial disease and should help to educate the public accordingly The taboo of leprosy is the cause of its spread in the East Leprosy is becoming a menace in India The clinician of today is equipped with the modern chemotherapeutic drugs to fight the scourge of leprosy About 2 per cent of all skin cases seen in India are cases of leprosy now

Etiology Leprosy is caused by an acid fast bacillus called Bacillus lepræ or Hansen s bacillus Hansen discovered the organism in 1874

Incubation period is not known May be from several month to year It is a low grade contagious disease in all the stages but at a certain stage of the disease it is infectious but at others it is not Nutrition plays a great part both in the spread of the disease and also in its effect on the system It is not a familial disease and a child separated after birth from a leprous mother may escape infection Upto the age of 3 years the possibility of getting the infection is maximum which gradually decreases with the growth of age People have also been found to get the infection at a late age probably by mucous membrane contact

Signs and Symtoms Classification (I) **Lepromatous type**—nodules are found all over the body On the face nodules (Fig No 94) appear which when coalesce gives a

bacilli with plasma cells, leucocytes, lymphocytes and histiocytes (Fig No 93)



Fig No 93
Histopathology of rhinoscleroma

Differential Diagonosis (1) Syphilis, (2) Leprosy,

Prognosis Is fair

Treatment Streptomycin is injected in gram 1 dose for 21 days with PAS 0 5 gm tablet—2 tablets 6 times daily after food Locally 10 p c PAS ointment

the body may be affected (Fig Nos 97 & 98) Lepra fever may also develop Nodules may appear on the

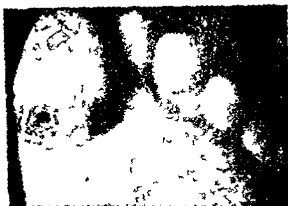


Fig No 96
Trophic ulcer
(On the planter surface of the great toe in a leper)

cornea destroying the eyes Lymphangitis *may occur* Claw hand may develop and is called **"leper claw"**



Fig No 97
Leprosy on left cheek
(Intermediate type)

lion-like face called **leonine facis** The ears and nose look larger in size Edema appears and fingers and toes

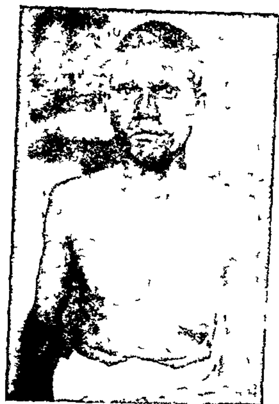


Fig No 94
Lepromatous leprosy showing unilatral gynecomastica in a male aged 35 years
(Case of Captain S N Roy)

look tense and separated Ulceration of fingers (Fig No 95) and toes and ulcers also appear under the soles when

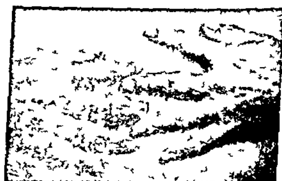


Fig No 95
Leprosy
(Tuberculoid type)

called **trophic ulcers** (Fig No 96) Skin of any part of

the presenting sign Thickening of the cutaneous nerve (Fig No 100) supplying the area is often the earliest sign found There may be anaesthesia or hyperesthesia Gynecomastica (Fig No 94) may develop in males which is unilateral or rarely bilateral



Fig No 99
Leprosy
(Tuberculoid type)



Fig No 100
Tuberculoid leprosy
(Thicked nerves behind left elbow)

The latest Indian classification of leprosy of 1955

(1) Lepromatous (L)
(2) Tuberculoid (T)
(3) Maculoanaesthetic (MA)
(4) Polyneuritic (P)
(5) Border line (B)
(6) Intermediate (I)

Thickening and abscess of nerves are usually seen Loss of hair, depigmention or pigmentation of the skin may be associated Epistaxis is a common symptom



Fig No 98
Leprosy of the Scrotum

(2) When the disease becomes chronic it is called the **Intermediate type** This type may develop also when a tuberculoid type of leprosy develops into the lepromatous type Lesions are erythemato papular Here *the acute symptoms and signs of lepromatous stage are* absent but merely exaggerated signs and symptoms than the tuberculoid type are seen This is also somewhat infectious

(3) **Tuberculoid type** *of leprosy is not infectious* It presents *varied clinical picture* Macular depigmented, hyperpigmented, erythematous, anhidrotic areas may be the only sign (Fig No 99) Macular depigmented patch with circumferentially hyperpigmented area also may be

the presenting sign Thickening of the cutaneous nerve (Fig No 100) supplying the area is often the earliest sign found There may be anaesthesia or hyperesthesia Gynecomastica (Fig No 94) may develop in males which is unilateral or rarely bilateral

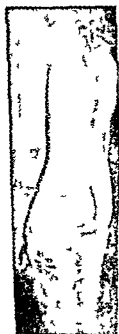


Fig No 99
Leprosy
(Tuberculoid type)



Fig No 100
Tuberculoid leprosy
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(6) Intermediate (I)

Diagnosis (1) Clinical examination for thickening of skin and nerves as well as depigmented and hyperpigmented areas of skin

(2) Pin prick and cotton wool touch test Sometimes instead of anaesthesia the patches may be hyperaesthetic Epicritic sensibility is first lost Temperature sense may disappear first hence the importance of testing with test tubes filled with cold and warm water In early tuberculoid type the loss of temperature sensation may be the only sign and in the fully developed tuberculoid type as well as in all the other types the sense of pin prick and cotton wool touch are lost Deep sensibility and gross pain sensations are also lost in the Intermediate and Lepromatous types of leprosy

(3) Skin scraping smear examination for Bacillus lepræ and staining with Zeil Neelsen stain for acid fast bacilli

(4) Nasal smear examination for acid fast staining

(5) Urine examination routine

6) E S R of fasting blood higher reading in leprosy moderately high (40 mm) in the intermediate and highest (70 mm or more in lapromatous type)

(7) Blood W R Pseudo positive

(8) Blood examination for total and differential count, Hæmoglobin p c and parasite

(9) Biopsy of the lesion and histopathological examination will show atrophy of the epidermis with Bacillus lepræ in the dermis is commonly seen in the

Lepromatous stage less commonly in the Intermediate stage and rarely in the Tuberculoid stage of leprosy Giant cells are seen in the Tuberculoid state Perineural infiltrate is also a feature of leprosy Destruction of the cutaneous nerve is commonly found in the tuberculoid type of leprosy Tuberculoma is seen in tuberculoid leprosy (Fig No 101) Perivascular infiltration of histiocytes lympho



Fig No 101
Section of Tuberculoid Leprosy showing tuberculoma
(Case of Major B Chukarbutty)

cytes and plasma cells in the upper part of dermis is characterstic and in the lepromatous stage the presence of large vacuolated histiocyte is called Lepra cell' or Globi body

Differential Diagnosis (1) Post Kala Azar dermal leishmaniasis (2) Syphilitic cutis (3) Vitiligo (4) Ringworm (5) Tuberculosis cutis, (6) Sarcoidosis (7) Pemphigus

Prognosis It is good with modern treatment

Treatment Improvement of the general nutritional stage of the patient is essential Patient should be

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2 to 5 years as need be The treatement is a prolonged one and the co operation of the patient is absolutely essential in the cure of the disease

Liver extract injection weekly are good adjuvant treatment during the one day rest period while on sulphone therapy

Isoniazide in dose of 200 mg by month in divided doses is given alone or with D D S for 6 to 12 months

Lepromatous reaction—Is characterized by exacerbation of the skin lesions and even there are new formation of lepra nodules in the eyes, all over the skin and viscera together with systemic reactions In this stage sulphone is useless Streptomycin or Dihydrostreptomycin in one gram dose is injected I M for 10 days

Sodium salt of chaulmoogra oil is injected intradermally and also I M weekly

Rehabilitation of the leprosy patients in the leprosorium is advocated Eradication of the disease is not difficult from a country but this is a problem of social medicine

TUBERCULOSIS

Tuberculous disease of the skin is becoming quite common in the tropics In one of the big cities *in India*, it has been estimated that out of all skin cases attending about 1 5 p c are tuberculous The approximate average number of tuberculous skin cases in India has been estimated to be about 2 0 *p c Either* the skin consciousness amongst the public or the increase in the incidence of this disease is responsible for this high percentage

given high protein diet with plenty of green vegetables and milk. Liver diet is preferable.

Medicinal. Sulphone therapy. If the blood examination shows no agranulocytosis and Hb p c is above 70 it should be started and can be given in all stages of leprosy. The blood examination should be repeated weekly as well as the examination of urine.

Novophone Y (Bengal Chemical), Sulphone (Cilag), Sisocarbazone (Albert David), Sulphetrone tablet (B W) or D A D P S (I C I) tablets are given as follows —

Started with ½ tablet once daily and is give for 6 days a week and no tablet on the 7th day ½ tablet twice daily for 6 days a week and repeat for 5 weeks. Patients who do not tolerate sulphone orally may be given intramuscular injection of 50 p c sulphetrone or Novotrone solution ½ to 1 c c weekly in first week then biweekly for 12 weeks. There are various other sulphone preparations available in the market. Diazone (Abbott) may be given as one tablet daily for a week then twice daily for 5 weeks followed by rest for a month. Repeat sulphones for 2 years with iron.

Patient should have rest for 4 weeks during which the examinations of E S R, skin snip smear for B Hansen and even biopsy should be repeated and during this time injections intradermaly and intramuscularly of Hydnocreol weekly are given or Streptomycin or Dihydrostreptomycin is injected I M daily for 10 days and repeating for 10 days after an interval of a day with P A S (4 grams T D).

After the examinations when results are negative and absence of bacillus the treatment should be continued for

Age Generally seen in the young age Children suffer mostly 80% of cases are seen under the age of 20 years But accidental inoculation with Koch s bacillus may occur at any age particularly in a family where there is present a case of open pulmonary tuberculosis Inoculation following vaccination or piercing of ear lobules of girls for putting on ornaments or after tattooing skin tuberculosis may develop at any age Postmortem wart on the fingers of doctors who are doing autopsy or are handling dead bodies in the anatomy hall develop warty tuberculous skin lesions on the fingers Warty lesions are also found on the sole after a thorn prick (Fig No 102



Fig No 102
Lupus verrucosum plantaris

is a problem to the specialists in skin disease in India at present A better dermatological knowledge amongst the *general practitioners in the tropics is certainly helping to* bring to light a larger number of cases of skin tuberculosis and is also helping in the treatment with the modern drugs

Definition Is a chronic granuloma of the skin due to *Bacillus* tuberculosis

Etiology Tuberculous disease *of the skin* is caused by Mycobacterium tuberculosis Koch described *four different types of bacilli human, bovine, avian and* piscine In India the human type is responsible for cent per cent cases of skin tuberculosis

Mycobacterium tuberculosis is a rod shaped, non motile, acid fast organism (A F B)

Tuberculosis is not a hereditary disease but chil dren born of tuberculous parents have susceptibility for this disease

Mode of infection

(1) Direct through the broken skin,

(2) Direct contact with an infected tuberculous *gland or a* sinus

(3) Auto infection such as from an open lesion either in the lungs or anywhere in the body,

(4) Hæmatogenous infection,

(5) *By extension peripherally,*

Sex Females are commonly effected In the tropics both the sexes are equally affected

discharging sinus Koch's bacilli may be cultured if pus is inoculated in a guinea pig in the laboratory and the ulcer



Fig No 103
Primary Tuberculous complex
(Case of Dr B N Banerji)

in the finger or toe shows clinical tuberculous granuloma which on pressing with a glass slide (Diascopy) shows yellowish granules (apple jelly nodules) Tuberculin test (Mantoux) is negative in the early stage and only becomes positive after 8 weeks There is no other tubercular focus anywhere in the body The skiagram of the chest may be negative in primary tuberculous complex and the Mantoux test positive Healing is by fibrous tissue formation and calcification with very little scar formation

(2) **Tuberculosis Verrucosum Cutis**—This is quite common and is often seen in hospital and in special practice Patients may be of any age but particularly common amongst children Hands and feet are the common sites Lesions are warty indurated, dry and found particularly on the palm sole (Fig No 102) and knuckles of fingers There is always present a history of injury or pin prick The Koch's bacillus enters through the

Pathology-After the Mycobacterium tuberculosis enters the tissues a non specific inflammatory reaction starts The introduction of living or dead tubercle bacilli or their disintegration produces a granulomatous lesion which shows histologically giant cells, epitheloid cells and lymphocytes Subsequently inocculation of the body with tubercle bacilli will produce a granuloma The formation of granuloma depends on the virulence of the organism and on the resistance of the tissues Allergic reaction to the Koch's bacilli and its toxin may take a great variety of forms The essential features of these reactions are that they are benign eruptions, symmetrical and often peripheral in distribution, showing a tuberculous histology without demonstrable presence of the organism and often occurring in tuberculous subjects These benign eruptions are called *tuberculids* Tuberculids are of various types Damage to the skin with tubercule bacillus is not so severe in the tropics and the incidence of cutaneous tuberculosis is much less on the pigmented skin in the tropical country

Classification Tuberculosis of the skin is mainly of two different types such as (A) Localized and (B) Hæmatogenous (A) Localized type may further be subdivided as follows

(1) **Tuberculous chancre**—which is the "**primary tuberculous complex**" It consists of two parts such as a primary tuberculous sore on the skin and a suppuration of the regional lymph glands (Fig No 103) It has been observed in children and in adults in the tropics There may be an injury on the finger or toe followed some times after by lymphadenitis The ulcer becomes indolent and the lymphadenitis suppurates and breaks leaving a

nose and the like Any other part of the body may be



Fig No 104
Lupus Vulgaris
(Lesions over the Knee joint and on in thigh look verucose)

effected (Fig No 106) with lupus vulgaris giving the typical diascopic appearance of apple jelly In debilitated subject the skin lesions break down (Fig No 107) and ulcer is formed Lupus vulgaris of soft parts like mouth and nose produces deformities and may take different forms such as pastular serpiginous and vegitative Lupus of the limbs (Fig No 108) may result in solid edema Lupus vulgari may affect other parts of the body also Tuberculin test is always positive in lupus vulgaris

(5) **Scrofuloderma**—is due to the local effect of the tubercle bacilli on the skin from some underlying tuberculous glands bones and joints Common sites

breach in the skin The infection is always produced by direct intracutaneous entrance of the bacillus Tuberculin reaction is always found positive Sometimes seen amongst medical practitioners and nurses The regional lymphadinitis may be present

(3) **Tuberculosis Cutis Orificialis**—the condition is found in patients with pulmonary tuberculosis Common sites of the ulcers are the muco cutaneous junction of lips and the tip of the tongue Indolent shallow ulcers may be found on the tip of the tongue but may also be present on the lips, anal region and on the glans penis Lesions may be nodular, ulcerated or papillomatous in types

(4) **Lupus Vulgaris**—is the commonest tuberculous skin disease and is easily diagnosed even by the busiest general practioner Inoculation lupus vulgaris is not uncommon Lupus vulgaris can develop in a person on the site of B C G vaccination 2 years after Tuberculous nodules develop and coalace to from the lesion of lupus vulgaris Some of the lesions become verrucous (Fig No 104) and remain small in size Any part the body may be affected but the commonest site is the face (Fig No 105) In the face the disease starts as a soft, *small, yellowish nodule near the nose and spreads very* slowly Sometimes it gets generalised and is called **"Lupus disseminatus"** If a glass slide is pressed on the skin (diascopy) yellow nodules are seen which is called apple-jelly nodule Soft shiny nodules spread like a *sheet peripherally and sometimes shows scales* also Lupus vulgaris may affect the whole face giving a dreadful appearance with drawing down of one corner of the upper or lower lip, ectropion, pinching of the

affected due to the dissemination of the organisms of pulmonary tuberculosis after measles and whooping



Fig No 166
Lupus vulgaris on the upper arm

cough The skin lesions are like pin head to half lentil in size Sometimes rash may be haemorrhagic also Bacilli are found all over the body Tuberculin test is negative

(2) **Tuberculosis miliaria disseminatus faciei**

are the neck (Fig No 109) and joints Commonly seen in young people The disease starts as a hard subcutaneous



Fig No 105
Lupus vulgaris of face

nodule which gets adherent to the overlying skin In time the skin ulcerates and an indolent ulcer or a fistula results Ulcers may be of different shapes but the edge is undermined and the base is fixed Cicatrix results on healing Scrofuloderma is in reality the cold abscess of the skin Tuberculin test is positive

(B) Haematogenous type of the tuberculous disease of the skin often starts from some internal tuberculous focus and is subdivided as follows

(1) **Acute miliary tuberculosis of the skin** is rare in the tropics Children are particularly

may occur on the face when it is called Acnitis Eruptions may occur on the hands when it is



Fig No 108
Lupus vulgaris of forearm

called Follicles Lesions are found on the trunk and lower limbs also (Fig No 110) Lesions start on the subcutaneous tissue and becomes pustular Central necrosis appears Lesions heal with a punched out scar

face is the only part of the body affected Lesions are



Fig No 107
Lupus vulgaris

papular and brownish in colour Papules are like half lentil in size The lesions come out in crops and may disappear spontaneously leaving pitted scars Diascopy shows apple jelly characteristic of the lesion Tuberculin *test is not helpful as it sometime gives* positive and at others negative reactions

(3) **Rosacea-like tuberculid**—these are reddish papular lesions half lentil in size appearing in large number and are distributed symmetrically over the cheeks and also on the forehead Lesions do not undergo necrosis Apple jelly characteristic is present on diascopy Commonly seen in women Takes a long time to heal Tuberculin reaction is positive

(4) **Papulo necrotic tuberculid** occurs at any age but is commonly seen in adults Affects both sexes It is a quite common skin condition in the tropics Eruptions

is commonly seen Lesions are painless and never itch Lesions are grouped and often appears in crops Tuberculin reaction is positive

(5) **Lichen scrofulosorum** is characterized by groups of pinhead sized papules without itching on the trunk and the extremities Involution takes place but relapse is quite common Tuberculin reaction is positive

(6) **Erythema nodosum** is charactrized by pea sized and little bigger sized dusky red nodules on lower extremities below the knees (Fig No 111) These are painful and non ulcerating in nature Tuberculin test may be negative



Fig No 111
Erythema Nodosum
(Case of
Dr B N Banerji)

(7) **Erythema indurata**—is a chronic recurring skin lesion of the legs of young people Commonly seen in girls just over teens and is seldom found in boys Distribution is bilateral and is situated on the calf muscles Subcutaneous erythematous nodules appear which coalesce and becomes a dusky red cutaneous nodule of about ½ to 1 inch in diameter These nodules may

and pigmentation Crusts form under which ulceration appears which heals with a scar Spontaneous healing



Fig No 109
Surofulodermia
Chin Strap type of
Lupus vulgaris



Fig No 110
Papulo necrotic tuberculid
(Case of Dr B N Banerjee)

stage of the disease cutaneous tuberculosis has no typical picture except that there is a nonspecific infiltration with polymorphonuclear leucocytes hystiocytes and tubercle bacilli may be found (b) In the advanced stage the epidermis shows no change but in the dermis there may be necrosis surrounded by epitheloid cells and giant cells (Fig No 112) (4) Guinea pig inoculation is done in the laboratories

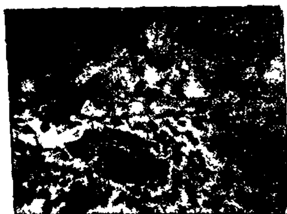


Fig No 112

Histopathology of Lupus Vulgaris showing giant cells and epitheloid cells

Differential Diagnosis (1) From dermal Leishmaniasis (a) Tropical residence (b) Skin snip smear for Leishman Donovan bodies is negative (c) Biopsy examination does not show Leishman Donovan bodies in the dermis (d) No history of K A and having had no antimony injection, (2) From cutaneous syphilis—particularly mistaken with nodulo cutaneous syphilide (a) Negative Kahn test, (b) Skiagram of bones does not show syphilitic changes, (c) Biopsy does not show

involute or ulcerate with necrosis of the subcutaneous tissue and the underlying tissue It is tender and painful Course is very chronic Tuberculin reaction is positive

Sign and symptoms Site is characteristic with a particular type of skin tuberculosis Lupus vulgaris commonly occurs on the face but may occur at other place as well Tuberculosis miliaria disseminata facie, Rosacea like tuberculid and acnitis occur on the face whereas papulo necrotic tuberculid occurs on the trunk and limbs Erythema induratum occurs on the calfs of both the legs Lichen scrofulosorum occurs on the trunk and on the extermities and verucosum cutis occurs on fingers toes, knees, elbows and on planter surface of feet Spread of the disease is very slow Tuberculous nodule formation is the primary skin lesion which when coalesce gives rise to different forms On pressure over the nodules with a glass (diascopy) yellow coloured granules (apple-jelly) are seen Tuberculous lesions heal by cicatrix formation It spreads centrifugally and heals at the centre by cicatrization Tuberculous lesions are very indolent and take a long time to heal

Complication (1) Eczema, (2) Impetigo, (3) Solid Oedema, (4) Cutaneous horn, (5) Carcinoma

Diagnosis (1) Clinical Examination, (2) Tuberculin Reaction test known as Montoux Test—intradermal injection is given with 0 1 c c of old tuberculin in dilution of 1 in 10,000 or 1,000 or 100 on one arm and with sterile distilled water on the other arm If positive, edematous flush appears within 48 hours Sometimes there is a central necrosis also This test is very helpful in the diagnosis of tuberculosis cutis, (3) Histopathology—(a) in the early

weekly or biweekly for a period of 6 to 9 months For a child the dose is reduced according to the age As a routine streptomycin daily or dihydrostreptomycin injections intramuscularly 1 gm per day for three weeks together with PAS 3 g 4 times a day by mouth for 2 months and then following up the patient with calciferol 50 000 i u twice daily by mouth for 6 months have been found useful in the tropics

Vitamin D2 is a useful drug but shortly after starting the treatment there is congestion in the skin lesions and also in any internal tuberculous focus This active reaction of the tuberculous focus may be fatal to the patient Impairment of the renal function may occur This Vitamin mobilises phosphorous and calcium Metastatic calcification of soft tissues may occur Improvement in lupus vulgaris with injections in the lesions of high potency Vitamin D2 at intervals of 2 to 3 weeks particularly in localized refractory lesions has been found helpful

Investigations necessary before calciferol therapy are (1) Skiagram of the chest to eliminate pulmonary tuberculosis, (II) Serum calcium and serum phosphorous level estimation (III) Examination of cardiovascular system, (IV) Blood urea estimation

Calciferol intoxication durig treatment should be watched which manifest as follows —

(1) General weakness fatigue loss of weight, (2) Gastrointestinal nausea and vomiting (3) Neuropsychiatric headache parasthesia, vertigo, (4) Hæmatological normocytic and hypochromic anæmia, (5) Urological

the presence of numerous plasma cells in the dermis and does not show thickening of the arteries, (3) From Leprosy (a) Impairment of sensation and nerve thickening are absent, (b) skin snip smear and nasal smear for B Hansen negative (c) Biopsy will not show B Hansen in the dermis No lepra cell is formed (5) From Lupus erythematosus (a) Distribution it not typical No atrophy of skin with peripheral pigmentation and scaling are seen, (b) Biopsy gives no characteristic picture of L E, (6) From Epithelioma histopathologically no characteristic of malignancy are seen, (7) From Acne Rosacea—histopathology is helpful, (8) Bromoderma and Iododerma histopathology is helpful, (9) Sarcoidosis—by histological examination and skiagram examination of chest, hands and feet, (10) Erythema Indurata from syphilitic gumma

Prognosis Is good with modern treatment in all cases of tuberculous disease of skin with few exceptions

Treatment (A) General treatment amounts to (1) Healthy surrounding with plenty of sunshine and fresh air (2) Good food rich in fat and vitamins and protein are also very essential Table salt is restricted

(B) Local treatment consists of (1) 50 p c P A S ointment as a dressing (2) Isonicotinic Acid Hydrazide ointment (1 in 100 INH) has been found to be effective (3) Ultra violet light exposure weekly is helpful

(C) Specific treatment consists of (1) Calciferol or Vitamin D2—this may be given orally or by injections in doses of 100 000 I U daily by mouth for a young adult otherwise healthy 600,000 I U injection intramuscularly

Calciferol may be used thereafter for a further period of 3 months If there is ulceration present 50 p c P A S ointment locally is indicated When sinus is present 20 p c P A S solution is introduced every day

Radiotherapy is also helpful in tubercular lymphadenitis in association with scrofuloderma of the neck or groin

SARCOID

Definition is a chronic granulomatous skin disease characterized by symmetrical plaque like nodular brownish skin lesions on the face and extensor surfaces of the limbs but bones and lungs, liver and lymph glands may be affected also

Etiology Cause is not known Bacillus tuberculosis is said to be the causative organism It is also thought to be a type of lymphoblastoma Is found 0 1 p c in tropical practice

Age No age is exempt Sex in both sexes

Types (1) Benign sarcoid of Boeck

(2) Lupus pernio of Besnier

(3) Subcutaneous sarcoid of Darier Roussy

Signs and symptoms Boeck s sarboid is red or brown half pea sized nodules developing on the face and the upper extremities which are distributed symmatrically Lupus purnio of Besnier is *manifested* by infiltrated brownish plaques and is symmetrically arranged Subcutaneous type of Darier Roussy type is characterized by the slow

albumin, R B C and cast in urine Slightly elevated alkaline phosphatase level and progressive nitrogen retention, (6) X ray shows particularly of soft tissue calcification, (7) Ophthalmological examination band Keratitis, (8) Uraemia, (9) Hypercalcemia

Streptomycin injection—1 gram daily injection I M for 3 weeks Dihydrostreptomycin in the same dose for the same period has been found equally useful in patients who do not tolerate streptomycin

Para Amino Salicylic Acid (P A S) Given by mouth in 3 gm dose 4 times a day for 3 weeks alone or together with streptomycin is useful 50 p c P A S ointment has been found very helpful in dressing tuberculous ulcers and 20 p c solution in dressing a tuberculous sinus

Isoniazid (I N H) 200 mg daily in divided doses by mouth is helpful particularly in cases which are resistant to calciferol therapy Isoniazid is one of the newer forms of treatment of lupus vulgaris As the tubercle bacilli acquire resistance to the drug, streptomycin is given to obviate that possibility and also to act as a synergist Isonicotinic acid hydrazide 300 mg by injection has been found to be very helpful even in the treatment of the primary tuberculous complex of the skin

In tuberculous skin disease the following method has been found to give very encouraging result Streptomycin 1 g by I M injection daily for 3 weeks together with by mouth P A S 3 g 4 times daily From the fourth week to 3 months Calciferol 50,000 i u by mouth twice daily and then Isoniazide 50 mg thrice daily for 3 months

liver extract is given by intramuscular injection in dose of 2 c c on alternate days and is sometimes helpful, (5) *Multivitamin is given by intramuscular injections*, (6) Calciferol may be given in dose of 50 000 I U twice daily for a long time keeping a check on blood calcium level, E S R and albumin and cast in urine, (7) Isoniazid in dose of 150 mg orally per day for a period of 3 months can also be used (8) Cortisone may be given orally starting with 25 mg every 6 hours in the first week and gradually reducing in about 6 weeks time (8) Cortisone may be locally infiltrated in the lesions, (9) *Hydrocortizone (Roussell) may be applied locally*

development of oval, skin coloured subcutaneous nodules over the trunk which are not symmetrically distributed

Besides the skin lessions there may be present lymphadenities with or without the mottling x ray change in the lungs and rarefaction and cyst-formation of the bones of the hands and feet Enlargement of the spleen and liver may be present together with enlargement of parotid glad, lacrimal gland, mammary gland and testis

Diagnosis (1) Symmetrical arrangement of skin coloured or brownish infiltered plaque or nodules on the face and trunk, (2) Biopsy-histopathology shows collections of epitheloid cells with few giant cells in the dermis, (3) Kveim test is positive is sarcoid, (4) Tuberculin test is negative

Differential diagnosis (1) Lupus vulgaris, (2) Lupus erythematosus, (3) Syphilitic cutis, (4) Leprosy, (5) Leukaemia cutis, (6) Eosinophilic granuloma of face, (7) Lymphocytoma of face

Prognosis Is good but sometimes patients develop tuberculosis and die

Treatment Prophylaxis is to avoid tuberculous infection or getting immunized with BCG vaccine

Curative (1) Arsenic should be given as Liq Arsenical in dose of m 3 in water thrice daily and for a long time may be continued also, (2) Calciferrol in dose 100,000 I U orally every day or 600 000 I U by intramuscular injection weekly may be tried, (3) Isoniazid has also been advocated in dose of 3 mg per Kilogram body wight for a period of 6 months, (4) Crude

pin head in size and is papular, (3) Blood picture shows slight leukopenia and increase in the number of mononuclear cell (4) Histopathology shows subcorneal vesicle formation and swelling of vascular endothelium in dermis

Differential diagnosis (1) Chicken pox (2) Small pox, (3) Urticaria, (4) Drug rash, (5) Pityriasis rosea, (6) Id reaction to fungus or bacteria

Prognosis Good in uncomplicated cases

Treatment Prophylaxis—Injection of (1) Convalescent serum (2) Pooled serum of several adults, (3) Anti measles serum (Bengal Immunity, Calcutta) (4) Gumma globulin in dose of O 1 c c per pound of body wieght may be given by intramuscular injection

Curative—Alkaline mixture with sedative To prevent complication Aureomycin or Terramycin may be used orally for 4 to 5 days or Penicillin crystalline G injections Locally liniment Calamine with 1 p c Hydrarg Ammon may be used Gumma globulin is given by intramuscular injection in dose four times the prophylactic dose

SMALL POX

This is also called *variola*

Definition Is an acute infectious and highly contagious skin disease characterized by bullous rash all over the body with grave systemic reaction

Etiology Is due to a virus Age—affects all ages but adults are commonly affected Sex—both sexes

Signs and symptoms Incubation period is 12 days Prodromal symptoms are headache, backache with high

CHAPTER IX
VIRUS DISEASES OF THF SKIN

Common viral skin diseases in the tropics are

1 Measles,	6 Herpes zoster,
2 Small pox,	7 Molluscum contagiosum,
3 Chicken pox,	8 Condylomata accuminata,
4 Herpes simplex,	9 Warts,
5 Herpes progenitalis,	10 Tropical bubo

Measles

Definition is an erythematous pin point to pin head sized lesion all over the body with systemic reaction and coryza affecting the infants and children

Etiology Virus is responsible Age Common in children and infants Sex—both the sexes suffer

Signs and symptoms Incubation period is 11 days There is always a prodromal symptom characterized by circumoral flush Later on pin point to pin head sized skin lesions appear all over the body with fever, conjunctivitis, rhinitis, bronchitis and inflammation of the buccal mucous membrane called Koplik s spot The rash is papular and is red in colour

Clinical types are —

(a) Mild type,
(b) Toxic type,
(c) Haemorrhagic type
(d) Congenital type,
(e) Pre natal type which is said to be responsible for the development of birth marks in the newborn

Diagnosis (1) Infants or children having coryza, bronchitis, conjunctivitis, rhinitis and Koplik's spot within the mouth, (2) Rash is red and pin-point to

Curative—Nothing is known Aureomycin or terramacin capsule 250 mg every 6 hours for 8 days together with vitamin C (200 mg) by mouth 4 times a day may be given Alkali mixture with a sedative is very valuable In the pustular stage and later on also lotio Coudy s bath twice daily followed by application of 2 p c Ung Hydrag Ammon is helpful For complications Penicilline crystalline G in 5 lacs dose is injected every day for 5 to 7 days Lesions before they are completly healed, should be touched with undiluted Coudy s lotion 4 times daily

CHICKEN POX

This is also called *varicella*

Definition Is an acute infectious skin disease characterized by the formation of vesicular rash and a mild systemic reaction

Etiology Is due to a virus The virus is said to be the same as the virus of herpes zoster

Age all ages but common in children Sex both sexes

Signs and symptoms Incubation period is 14 days There is no prodromal symptom During the first three days vesicular rash appears on the head face and the trunk and then spreads down the extremities Rashes come out in successive crops The vesicles rupture and crust forms in 10 days Itching is a prominent symptom Scratching gives rise to secondary infection Constitutional symptom is either absent or very mild

Diagnosis (1) Vesicular rash developing on head face or trunk with mild constitutional symptoms (2)

fever By the fourth day the temperature falls and the symptoms disappear but reddish papular rash comes out The papules soon become vesicular and after four days become pustular Site of the rash is the face and the upper part of the body and the upper extremities Lesions may appear on the mucous membrane and conjunctivae with a red halo round each vesicle The second rise of temperature with toxicity again appears The pustule has a thick wall The lesion may be discrete or may be confluent in nature The pustular lesion soon becomes umbilicated The lesions scale off from the eleventh day leaving pitted scars called "pock mark" Pain all over the body and restlessness are the prominent symptoms *Sometimes* the vesicle may be filled with blood when it is called *haemarrhagic small pox* and is a grave condition

Diagnosis (1) Prodromal symptoms, (2) Second rise of temperature when the rash becomes pustular with toxic symptoms, (3) Histopathology shows multinucleated vesicle with ballooning degeneration of cells in the stratum mucosum

Differential diagnosis (1) Drug Rash (2) Chicken pox

Chicken pox	Small pox
1 Prodromal symptoms—Nil	1 Preset
2 Appearance—with rise of temp	2 after temperature falls
3 Lesions—vesicles are superficial	3 Vesicles are deep seated
4 Distribution of rash—head face and body	4 Face and expo ed parts

Prognosis Is grave in non-vaccinated cases than in vaccinated cases

Treatment Prophylaxis—pox vaccination gives a long lasting immunity Repetition of vaccination is necessary during an epidemic

symptoms, (4) May be associated with pneumonia or malaria and (5) Histopathology—In the epidermis vesicle is formed in the stratum mucosum due to coagulation necrosis In the dermis there is oedema with dilatation of blood vessels and perivascular infiltration

Differential diagnosis

(1) From Herpes Zoster

Herpes Simplex	Herpes Zoster
1 Does not follow nerve routes,	1 Develops along nerve routes,
2 Recurrent	2 Non recurrent,
3 Painless	3 Painful,
4 Leaves no scar	4 Leaves scar,
5 No post herpetic neuralgia,	5 Post herpetic neuralgia often results

(2) From Chancroidal ulcer of genitalia by the history of exposure Ito Reinsterna s test and by the presence of B ducrey on smear examination

(3) From Syphilitic ulcer by the history of exposure, presence of T pallidum by the dark field examination of smear and Blood Kahn and W R tests

Prognosis good

Treatment Touch several times during the day and night with spirit rectificatus and dress with dusting powder where possible May be sealed with collodion When vesicles have ruptured 1 p c Ung Hydrag Ammon locally is helpful Repeated vaccination with small pox vaccine is of great value in recurrent herpes simplex Aureomycin may be used as ointment and orally as capsule 250 mg every 6 hours for 5 days

Vesicle is mutiloculated and is found in the stratum mucosum Intranuclear inclusion bodies called "Lipschutz bodies" and ballooning degenertion of cells of stratum mucosum is found

Prognosis Good

Treatment Prophylaxis avoid contact with a patient of varicella or herpes zoster

Curative-Aureomycin or terramycin capsule (250 mg) by mouth every 6 hours for 8 days together with Elixir Vitamin B Complex may help Locally lotio Condy's bath followed by 2 p c Ung Hydrag Ammon application

HERPES SIMPLEX

Difinition Is an acute or chronic skin disease characterized by vesicle formation anywhere on the skin or mucous membrane

Etiology Is caused by a virus Is found associated with cold, pneumonia and malaria Virus may remain latent for many years and may get reactivated by trauma

Signs and symptoms Grouped vesicles which are pin-head or larger in size are found on slightly red skin Common sites are face and the genitals but may be found anywhere on the body Occur also on the mucous membrane of mouth May rupture forming crusts and getting well without leaving scar in a week s time May recur after sometimes In patients with atopic eczema herpes simplex virus causes an extensive vesicular disease with high fever and lymphadenitis when it is called *Kaposi's varicellifrom eruption*

Diagnosis (1) Grouped vesicles on slightly red skin, (2) Recurrent nature of the lesion, (3) No constitutional

HERPES PROGENITALIS

Definition Is a skin disease characterised by the development of recurrent grouped vesicles on the body of the glans penis and also on the vulva

Etiology Is due to a virus

Age—adults are affected Sex males commonly

Signs and symptoms Several tiny vesicles with erythematous rings appear in crops on the penis and over the glans penis in males and upon the labia majora and minora vesitbule and perinium of females It is painful and causes burning sensation When vesicles rupture undermined ulcers appear

Diagnosis Recurrent grouped vesicles on the genitalia of adults

Differential diagnosis (1) Chancroid, (2) Multiple hard chancre, (3) Fixed drug rash

Prognosis Good

Treatment Dusting powder is helpful consisting of calomel and zinc oxide in equal parts 3 p c Aureomycin ointment is also advocated

HERPES ZOSTER

Difinition This is an acute skin disease charac terised by pain fever and vesicle formation on one side of the body

Etiology Infection of the posterior nerve root ganglion by a virus and is supposed to be the neurotro pic strain of small pox virus May occur at any age

KAPOSI'S VARICELLIFORM ERUPTION

Definition Is an acute contagious, bullous disease with fever of infants due to a virus

Etiology It is due to the herpes simplex virus

Age—children are affected specially but adults are rarely affected People with *eczema suffer usually*

Signs and symptoms The disease occurs suddenly Grouped bullous lesions appear on the face with fever The lesions may then spread on the scalp and down the neck and appear *in sucussive crops* Lymphadenitis also occurs Sometimes lesions are umbilicated Lesion undergoes involution, crust forms and exfoliates *Diarrhoea may occur Anuria also develops Otitis* media and corneal ulcer also develop

Diagnosis (1) Sudden appearance of grouped vesicles on face with fever in a patient suffering from some skin disease, (2) examination of blood shows leucopenia, (3) histopathology shows intraepidermal vesicle formation which later becomes subepidermal Nuclear inclusions are present Infiltration in the dermis is dense

Prognosis Is good

Treatment Prophylaxis is that a patient of skin disease should not be vaccinated nor should come in contact with a patient of herpes simplex

Curative is to give sulphadiazine tablet one every 6 hours for 5 days with Mist Alkali, Penicilline crystal line 'G' is also injected I M in O 2 Mega unit twice daily for 5 days Vitamin C (200 mg) given orally 4 times a day Locally *Liniment* Calamine with 1 p c phenol is advocated and 2 p c Ung Hydrag Ammon at night

rarely get pain Small vesicles appear (Fig No 113) for a week on red patches of the skin along the course of a nerve and crusts form May be on the trunk or on the limbs There may be successive crops (Fig No 114) The course is about 2½ weeks In the herpes zoster of face the first branch of the sensory division of the trigeminal nerve is usually affected and may result also in corneal ulceration

Scars are either hyperpigmented or depigmented It is not recurrent The bulla fluid is serous to start with becoming purulent later on but sometimes may be haemorrhagic also Neuralgic pain usually persists for a long time Idiopathic herpes occurs without any cause

Types of herpes zoster are (i) Herpes frontales, (ii) Herpes ophthalmicus, (iii) Herpes facialis (iv) Herpes pectoralis (v) Harpes abdominis, (vi) Idiopathic herpes

Diagnosis (i) Unilateral painful distribution of hyperaemia or vesicle (2) Grouped vesicle on red skin (3) Fever and malaise (4) Non recurrent, (5) Course about 2½ weeks, (6) Heals leaving pigmentation or depigmentation at the site of the lesions, (7) Histopathology shows in the epidermis a vesicle formation due to ballooning degeneration and intranuclear inclusion bodies are *formed* Bulla is formed *in* the stratum mucosum In the dermis there is *oedema* with dilatation of vessels and cellular infiltration Virus also causes inflammation and even *necrosis* of the posterior spinal root ganglion of the sensory nerve

Differential diagnosis (1) Herpes simplex, (2) Varicella

both sexes May be seen associated with arsenic therap and leukaemia cutis



Fig No 113
Herpes Zoster
(Case of Captain S N Roy)

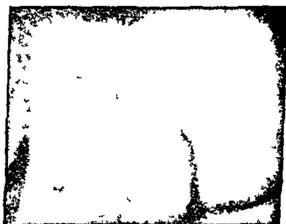


Fig No 114
Herpes Zoster
(On the sacral region and
thigh of a child aged 6 years)

Signs and symptoms unilateral intercostal redness with hyperaesthesia may be the earliest manifestation or there may be fever with pain on the part Children

rarely get pain Small vesicles appear (Fig No 113) for a week on red patches of the skin along the course of a nerve and crusts form May be on the trunk or on the limbs There may be successive crops (Fig No 114) The course is about 2½ weeks In the herpes zoster of face the first branch of the sensory division of the trigeminal verve is usually affected and may result also in corneal ulceration

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Differential diagnosis (1) Herpes simplex, (2) Varicella

Prognosis Usually good but sometimes may destory the eye or lesions may become gangrenous when the prognosis is grave

Treatment (1) Pain is relieved by Aspirin or by Potassium Bromide with Sodium Salicylate, (2) Pituitory extract O 5 to 1 O c c I M injection once daily *for 2 to 3 successive days before the* development of vesicles aborts or (3) Dihydroergotamin (Sandoz) is injected I M in 2 c c dose twice daily for 2 to 3 days, (4) Antibiotic may be used—Aureomycin *250 mg capsule by mouth with water 6 hourly for 4* days, have proved of great value During aureomycin therapy, vitamin-B complex should be administered also 4 times daily and thereafter also for a week *thrice daily after food*, (5) Vitamin B^{12} in large *doses* (500 microgram daily) is helpful to allay the post herpetic neuralgia when injected intramuscularly for two weeks, (6) Cortisone Roussell may be given in severe cases and particularly *in old patients The dose should be 25 mg by mouth* every 6 hours for 3 days with vitamin B complex and vitamin C for the first week, 12 5 mg with vitamin B complex and vitamin C every 6 hours for the next *3 days, and then 5 mg 6 hourly for 3 days, 5 mg 8 hourly for a day and then 12 hourly* on the last day, (7) Locally Lotio Calamine with 1 p c Phenol application every hour is advocated, (8) X ray application to the posterior nerve root ganglion *of the spinal cord is helpful* Ultra Violet exposure is sometimes helps

MOLLUSCUM CONTAGIOSUM

Etiology It is a virus disease and is mildly contagious Commonly seen in children Incubation period

is several month Brick shaped virus particle of molluscum' contagiosum has been demonstrated by electronic microscope



Fig No 115
Molluscum contagiosum
(On the umbilicus)

Signs and symptoms Pin head to pea sized nodular growth on the healthy skin The nodule has an umbilication at its top It has a pearly appearnce Grows very slowly Common sites are face hands, chest, back adomen (Fig No 115) and may be on genitalia (Fig No 116) Fresh lesions develop on a scratch mark (Koebner s phenomenon positive) Lesions are neither painful nor itchy

Diagnosis (1) Pearly tumour with umbilication, (2) Commonly in the pediatric age (3) Histopathology-multiple bodies grow down from the epidermis There

is degeneration of the cells of the stratum corneum forming a homogeneous eosinophilic inclusion body called "Molluscum body" Dyskeratosis is a feature



Fig No 116
Molluscum Contagiosum on the genitalia of an infant
(Case of Major B Chakrabutty)

Differential Diagnosis (1) Infective wart and (2) Epithelioma

Prognosis Good

Treatment Locally each tumour should be picked off with a toothed desecting forceps and the base is cauterized with phenol Electro cautery can also be used 50 p c Trichloracetic acid application after puncturing is helpful Antibiotic (Aureomycin 250 mg orally 6 hourly for 5 days with Vit B Complex orally) has definite value in molluscum contagiosum Sulphapyridine in dose of O 5 gm orally every 4 hours for 5 days is also advocated

CONDYLOMA ACCUMINATA

Definition It is a skin disease characterised by the formation of a collection of thin and elongated warty growths due to a virus

Etiology It is due to a virus and is predisposed by an irritating discharge

Age—found in adults Sex—seen in both sexes

Signs and symptoms It is associated with gonorrhoeal discharge in males and vaginal discharge in females Pregnancy is also sometimes associated in in famales Warty growths like cauliflower is found around the vulva, around the corona glandis and sometimes in the crural regions (Fig No 117) The colour of the warts are greyish yellow



Fig No 117
Condyloma accuminata

Diagnosis (1) Cauliflower like growth around the vulva, corona glandis, crural region in adults, (2) Histopathology shows dilatation of vessels in the dermis with acanthosis of the stratum mucosum of epidermis

Differential diagnosis (1) Condyloma lata, (2) Cancer

Prognosis Good

Treatment Prophylaxis is to keep parts clean and daily bath Curative is to paint the lesion with 20 p c

podophyllum resin in liquid paraffin protecting the skin and washing it Surgical removal with a diathermy needle may be done

WART

It is also known a *Verruca* or *Infective wart*

Etiology Is caused by a virus

Signs and symptoms Classification (i) Verruca vulgaris, (ii) Verruca digitata, (iii) Verruca filiformis, (iv) Verruca plantaris, (v) Verruca plana, (vi) Condylomata acuminatum

Warts are skin coloured, circumscribed papillary growth, May occur on the skin all over the body (Fig No 118) There is no pain or tenderness



Fig No 118
Infective warts
(Case of Dr K C Kandhari)

except in verruca plantaris where pain is felt on walking Infective wart is commonly seen on the face of children

Diagnosis (1) Skin coloured papillary tumour (2) Histopathology shows hyperkeratosis, acanthosis and elongation of the rete ridges

Differential diagnosis (1) Molluscum contagiosum

Treatment Removal of the warts by electro cautery and Carbon dioxide freezing Sometimes repeated daily applications of 20 p c Acid Salicylic in Collodion can cure the condition

TROPICAL BUBO

This is also known as *lymphopathia venereum* or *lymhogranuloma venereum*

Definition Tropical bubo is a venereal disease caused by a virus and is characterized by a genital sore followed by the enlargement of inguinal glands

Etiology This is due to a virus infection acquired by sexual contact but rarely even by extra genital contac Incubation period is 3 weeks May affect both sexes Age—adults are the victims

Signs and symptoms After sexual contact grouped vesicles appear on the mucous membrane of the genitalia They are painless and discharge colourless serous fluid During this time there may be fever with anorexia Within a week the lesions heal without scar formation 2 weeks after the appearance of the genital lesions the inguinal lymph glands of one side only get enlarged and tender Later on the lymph glands become fixed (Fig No 119) to the skin and get slowly enlarged The skin over the gland becomes red and sinuses

are formed through which seropurulent fluid comes out The condition may heal after several months leaving elephantiasis of the genitalia or the patient may develop virus meningitis or meningo encephalitis, polyarthritis and rectal strictures may also develop



Fig No 119
Tropical Bubo
Lymphogranuloma venereum
(Case of Dr B N Banerji)

Diagnosis (1) History of sexual contact followed by herpetic lesion on the genitals accompanied with fever and malaise and later on unilateral inguinal lymphadinitis, sinuses and elephantiasis of the genitalia, (2) Frei's test positive, (3) Biopsy—histopathology shows dense infiltration with plasma cells in the dermis together with epitheloid cells and giant cells

Differential diagnosis Granuloma venereum

Prognosis Is fair these days with modern treatment

Treatment Sulphapyridin for 10 days, Aureomycin for 3 to 6 weeks with Vitamin B complex, Streptomycin for 6 weaks, Anthiomeline 20 injections Superficial X ray therapy may be tried Surgical excision is often helpful

CHAPTER X

PARASITIC DISEASES OF THE SKIN

The common parasitic diseases found in the tropical dermatological practic are mainly

(A) Vegetable parasites viz fungus, (B) Animal parasites viz (i Scabies and (ii) Pediculosis

The common vegetable parasitic infections in general practice in the tropics are those due to Tinea and Actinomycosis

Tinea affects the skin, nail and hair There are three common types of fungi such as (1) Microsporon, (2) Eqidermophyton and (3) Trichophyton

The disease is called ringworm infection, or fungus infection or Tinea infection

Tinea infection can be classified as follows
(1) Tinea capitis (2) Tinea barbae (3) Tinea corporis (4) Tinea cruris, (5) Tinea pedis and Tinea manuum (6) Tinea versicolour (7) Erythrasma, (8) Favus and (9) Trichonocardiosis axillaris

TINEA CAPITIS

Tinea capitis is also known as ringworm of the scalp Seen commonly amongst prepubertal children but is rare in India Cases are seen in India only in the hills of Darjeeling, Simla and the like When rarely occurs it is seen in the epidemic form in the schools

are formed through which seropurulent fluid comes out The condition may heal after several months leaving elephantiasis of the genitalia or the patient may develop virus meningitis or meningo encephalitis, polyarthritis and rectal strictures may also develop



Fig No 119
Tropical Bubo
Lymphogranuloma venereum
(Case of Dr B N Banerji)

Diagnosis (1) History of sexual contact followed by herpetic lesion on the genitals accompanied with fever and malaise and later on unilateral inguinal lymphadinitis, sinuses and elephantiasis of the genitalia, (2) Frei's test positive, (3) Biopsy—histopathology shows dense infiltration with plasma cells in the dermis together with epitheloid cells and giant cells

Differential diagnosis *Granuloma venereum*

Prognosis Is fair these days with modern treatment

Treatment Sulphapyridin for 10 days, Aureomycin for 3 to 6 weeks with Vitamin B complex, Streptomycin for 6 weaks, Anthiomeline 20 injections Superficial X ray therapy may be tried Surgical excision is often helpful

as it destroys all the hair follicles Itching is one of the symptoms



Fig No 121
Ringworm of Scalp
(Kerion type in a boy aged 6 years)

Diagnosis (1) Wood's light examination—Wood's light is the filtered ultraviolet radiation When the head is examined in a dark room with Wood's light the infected hairs show fluorescence Green with Microsporon audouini and Microsporn canis but bluish with Trichophyton (2) Microscopical examination of infected hair under microscope for fungus (3) Culture of the infected hair for fungus

Differential dignosis (1) Alopecia areata, (2) Trichotillomania (3) Syphilitic alopecia, (4) Seborrhoic capitis and (5) Psoriasis of the scalp

Prognosis Is good except in acute type of Microsporon canis infection because it develop Kerion celsi which results in permanent baldness

Treatment Prophylactic—the patient should not be allowed to go to the school and should protect his head with a cap which should be boiled daily to prevent the spread of infection

Locally (1) Shaving the head should be done for children followed by (2) Washing the head with soap

Common organism is the *Microsporum audouini* which is very rare in India particularly in the plains *Microsporum canis* is sometimes found both in adults and children only in those who are found closely associated with dogs and cats Sometimes *Trichophyton violaceum* is also found responsible which produces black dot lesion

Signs and symptoms Starts as a scaly patch on the scalp (Fig No 120) where the Microsporum canis infection is present and the condition becomes acute

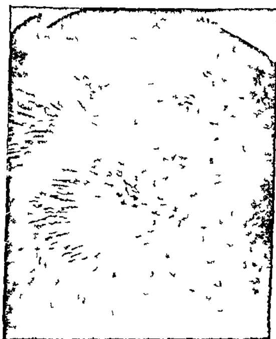


Fig No 120
Tinea Capitis
(On the scalp of a girl aged 4 years)

A boggy swelling develops on the head (Fig No 121) with multiple openings called *Kerion celsi* When it gets well permanent baldness is left due to *Kerion celsi*

Differential diagnosis (1) Contact dermatitis, (2) Bacterial folliculitis, (3) Drug rash (Iodide or Bromide)

Treatment In the acute stage 1 p c Lotio Ichthyol application is helpful During the subacute and chronic stages 4 p c Ung Hydrarg Ammon application or Ung Whitfield locally are helpful

TINEA CORPORIS

Tinea corporis produces ringed lesions of erythemato squamous type or only squamous type of lesions are found on the body (Fig No 123 & 124) Causative fungus are Trichophyton and Microsporon Attacks all ages and both sexes Itching is annoying Itching results



Fig No 123
Tinea Corporis



Fig No 124
Tinea Corporis et Cruris

and water every day, (3) Application of an ointment (Containing Hydiarg Ammon —gr XX in Vaseline—oz 1) after bath and before going to bed at night

After shaving and washing the head with a bland soap and water application twice daily may be applied (1) Ung Whitfield (Acid Benzoic gr 25, Acid Salicylic gr 15, Vaselin alba oz 1), (2) Sodium propionate or (3) Undecylenic acid

TINEA BARBÆ

Tinea barbæ is known as the ringworm of the beard Tinea barbæ is the chronic ringworm infection of the hairy part of the beard region (Fig No 122) Both microspora and trichophyta are causative organisms



Fig No 122
Tinea barbae
(Kerion type)

Tinea barbæ may be (1) Ringed type, like a coin in shape where there are broken hairs with a patch of eczema or (2) inflammatory type which may be pustular or kerion celsi type or may be (3) sycotic type with crusting and broken hairs

Diagnosis (1) Microscopical examination of an infected hair, (2) Culture from the lesion, (3) Wood's light examination is also very helpful

the fungus infection of the groin perineum and upper part of the thighs The causative fungus is the Epidermophyton inguinale The lesion produced is commonly a bilateral flexural infective eczema The margin is clear cut with erythematosquamous lesion and is somewhat raised from the surface of the skin (Fig No 124) The itching is very severe There may be associated Id reaction all over the body Commonly seen in adults of both sexes

Diagnosis (1) Skin scraping examination shows the presence of fungus (Fig No 125), (2) Culture of the skin scraping for fungus

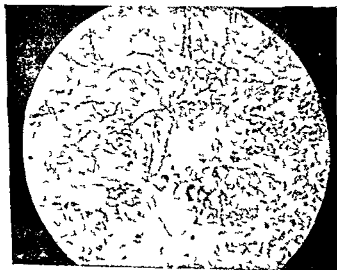


Fig No 125
Microscopic view of fungus
from the skin scraping
(Case of Dr G Tanja)

Differential diagnosis (1) Moniliasis, (2) Folliculitis

in secondary infection with strepto and staphylococci Sometimes multiple gyrate type of lesions appear with granulomatus margin Typical ringed lesion is sometimes found on the body (Fig No 123)

Diagnosis (1) Skin scraping is to be examined on a glass slide with 2 to 3 drops of 10 p c Sodium Hydroxide and covering with a cover slip The slide is warmed and examined under microscope, (2) Culture of the skin scraping for fungus

Differential diagnosis (1) Eczema, (2) Seborrhoic darmatitis, (3) Syphilitic cutis, (4) Leprosy, (5) Psoriasis

Prognosis is good

Treatment Hygiene is very important The patient should use a bland soap like Margo Soap (Calcutta Chemical) and all the clothings and towels used by the patient should be sterilized daily by boiling for at least one week during the course of treatment Application locally of Ung Whitfield regularly or Undecylenic acid are helpful When there is a localized patch present painting the part with Tn Iodine twice daily or 2 to 5 p c Ung Chrysophanic acid, 2 to 5 p c Ung Derobin or 2 to 5 p c Ung Cignolin are very helpful A check should be kept on the urine for albuminuria Ween local application of these medicines produces sensitization the local application should be stopped and Lotio Calamine is to be applied locally for 7 days before treatment is resumed

TINEA CRURIS

This is also known as *Dhobi's itch* Tinea cruris is

From this place it extends downwards and all the interdigital spaces are involved and gives the typical look of 'Haja There is sodden skin present in the interphalangeal spaces Similarly in the hand one of the interdigital spaces are involved first and all others are ultimately affected This is the common type found in maidservants and in those who keep their hands and feet wet (2) Another type of lesion is the vesiculobullous type and involves the plams and soles without affecting the interdigital spaces, (3) A type of infection where the skin of the soles become thickened (Fig No 127) (4) Sometimes nails of fingers and toes are affected



Fig No 127
Tinea plantaris
(Case of Dr G Panja)

(Fig No 128) which become black, thickened and furrowed

Sometimes the whole palm is involved and becomes chronic with desquamating lesions Rarely erythematosquamous lesions persist on the palm or sole

Treatment Prophylaxis is the scrupulous hygiene In treating a patient the hygiene must be followed rigidly Patient's underwear and towels should be boiled every day after bath In the oozing stage treat as any eczema that is if the oozing is profuse 1 p c lotio silver nitrate soaks every hour for 24 to 48 hours Next 1 p c aquous lotio gentian violet painting followed by Ung Whitfield application for 2 weeks In intractable cases 1 to 2 p c Derobin or Cignolin in Acetone may be painted Undecylenic ointment may be used with advantage

TINEA PEDIS

Tinea pedis and Tinea manuum is commonly known as Athlete s foot or *Epidermaphytosis* This is the ring worm infection of feet and hands 'Haja' or '*Paulagna*' are common terms in Bengal and Bihar for this condition

Trichophyton rubrum is the common fungus but Trichophyton interdigitale is also found



Fig No 126
Tinea Pedis
(Fungus infection in b tween toes and Lichen simplex on ankle)

(1) Commonest type the commonest site is the fourth interdigital space of one or both feet (Fig No 126)

From this place it extends downwards and all the interdigital spaces are involved and gives the typical look of Haja' There is sodden skin present in the interphalangeal spaces Similarly in the hand one of the interdigital spaces are involved first and all others are ultimately affected This is the common type found in maidservants and in those who keep their hands and feet wet (2) Another type of lesion is the vesiculobullous type and involves the plams and soles without affecting the interdigital spaces, (3) A type of infection where the skin of the soles become thickened (Fig No 127) (4) Sometimes nails of fingers and toes are affected



Fig No 127
Tinea plantaris
(Case of Dr G Panja)

(Fig No 128) which become black thickened and furrowed

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Fig No 126
Tinea Pedis
(Fungus infection in between toes and Lichen simplex on ankle)

(1) Commonest type the commonest site is the fourth interdigital space of one or both feet (Fig No 126)

The hygiene of the feet and hands should be carefully followed. Daily washing with soap and water followed by the application of medicines and the patient should boil the shocks, gloves and towels daily. Patient should better use a separate bathroom.

'ID' REACTION

'Id Reaction is an allergic reaction to the infection by fungus or bacteria. Together with ringworm infection there may be lichenoid rash all over the body and is known as trichophitid (Fig No 129) reaction.



Fig No 129
Trichophytid
(Id reactin to fungus).

Trichophitid may be sudden and may appear even during the course of treatment. Itching may be present. Fungal diseases have a tendency to hypersensitivity which is an allergic state and purduces superficial inflammation all over the body.

Diagnosis (1) Skin scraping for fungus, (2) Culture of skin scraping for fungus



Fig No 128
Tinea Unguam

Differential diagnosis (1) Dermatitis venenata or contact dermatitis, (2) Drug rash, (3) Papulosquamous syphilis of palm and sole, (4) Acrodermatitis perstans tropicalis

Prognosis Good

Treatment In the acute stage it is to be treated with 1 p c aquous lotio Silver Nitrate soaks for *2 to 3* days When the oozing subsides treat with painting of 1 p c aquous gentian violet followed by Ung Whitfield application and the parts to be kept covered by bandaging or by wearing shocks and gloves *To be* repeated twice daily for a period of two weeks In the chronic stage one of the following may be used (1) Ung Whitfield, (2) 2 p c Ung Derobin or Cignolin, (3) Ung Zinc Undecylenate, or (4) Castellani's Fuschin paint

Diagnosis (1) Skin scraping when seen with 10 p c Sodium Hydroxide under a microscope Mala furfur is seen in the scraping (2) Culture of skin scraping

Differential diagnosis (1) Depigmented stage of dermal leishmaniasis, (2) Seborrhoea, (3) Ringworm and (4) Leprosy

Treatment Improvement of the general health is essential Hygiene is important in the form of boiling of the underwears and towels daily Locally applications of one of the following —

Ung Whitfield 5 c c Ung Sulphuris, local application of 25 p c Sodium hyposulphite aquous solution followed immediately after with 10 p c aquous Tartaric acid solution Sometimes application of Tn Iodi mitis followed by 1 p c aquous silver nitrate is helpful

ERYTHRASMA

Erythrasma is a noninflammatory fungus infection of the type known as *Nocardia* Common sites are axillæ and crural regions Starts as a macular, reddish, circular spot which may be as large as a coin Gradually the colour changes to yellow

Diagnosis Skin scraping shows Nocardia

Differential diagnosis (1) Tinea versicolor and (2) Tinea cruris

Treatment Shaving the hairs and keeping the part clean with soap and water washing are essential followed by daily application of Tn Iodi mitis application or Ung Undecylenic acid or 10 p c alcoholic solution of sodium propionate Hygiene shold be follwed

The 'Id' reaction is caused by the absorption of the fungal toxin in the blood from the site of the lesion which circulates in the blood and comes out in the form of a rash only when the skin is sensitized

Diagnosis (1) Associated with the 'id' reaction there should be present ringworm lesion on the body, (2) Skin scraping from the ringworm lesion for fungus is positive, (3) Culture of the scraping from the ringworm lesion is positive and (4) Fungus is absent in 'Id' lesion

Differential diagnosis (1) Early secondary syphilis, (2) Drug rash, (3) Pityriasis rosea and (4) Seborrhoea

Treatment (1) Locally lotio Calamine soaking for 2 to 6 days followed by 1 p c Ung Acid salicylic application, (2) Antihistaminic drugs are helpful when given in the form of one tablet or capsule (Antistin, Benadryl, Calcilusin or Sandostin) three times daily for 5 days, (3) Vitamin C (200 mg) tablet 6 hourly for 1 week is also helpful

PITYRIASIS VERSICOLOR

Definition Tinea versicolor is caused by a fungus

Etiology Is due to a fungus called *Malassazia furfur* In the tropics Tinea versicolour and Tinea nigra are due to the same organism

Signs and symptoms Is seen in all ages and in both sexes Light brown coloured macular spots which coalesce to form nummular patches or very big sheets Commonly found on the V of the neck like a Japanese fan May also be seen on face, axilla, neck, chest, back and thighs Scales can be seen with a hand lens Itching is present which may be quite severe at times

Diagnosis (1) Scutula and mousy odour with alopecia of the scalp (2) Wood's light is helpful

Differential diagnosis (1) Tinea capitis (2) Seborrhoic eczema (3) Lupus Erythematosus

Prognosis Alopecia when produced is permanent

Treatment (1) Hygiene of the scalp, (2) Locally Ung Whitfield or Ung undecelynic acid applications

ACTINOMYCOSIS

Actinomycosis is not a rare disease in the tropics A type which commonly affects the sole is prevalent in South India and is known as *Actinomycosis madurae*

Signs and symptoms The infection starts as a small sore and then an indolent growth develops which is riddled with sinuses Serous discharge with yellow granular bodies exude The granules when pressed with a cover slip on a slide and are seen under microscope shows Ray fungus In culture actinomycosis grows

Differential diagnosis (1) Syphilis, (2) Cancer

Prognosis Should be guarded as it may involve muscles, bones and even internal organ and can cause death In an early case the prognosis is fair

Treatment Prophylaxis is to put on foot wear Curative Penicillin cyrstalline 'G 0 5 Mega unit is injected I M daily for 3 to 6 weeks Streptomycin in gram dose may be injected daily for 4 to 6 weeks with PAS orally Aureomycin (250 mg) capsules 6 hourly with Vit B complex 3 to 6 weeks Iodide should be given by mouth for a long time in high dose

FAVUS

Definition Is a fungus disease caused by Trichophyton Schoenleini which affects the scalp primarily and also the body

Etiology Both sexes are equally affected

Signs and symptoms Commonly affects the scalp but rarely other parts of the body and the nail Starts as yellow vesicles Crust forms which is cup shaped called the *scutulum* leaving a depressed scar Hair is irregularly affected and grows along the length of the hair It is a very chronic disease It produces permanent alopecia Patients emit a mousy odour Both the hair and the skin of the scalp (Fig No 130) are involved



Fig No 130
Favus of Scalp

Head looks moth eaten The body may show erythemato follicular lesion Affects both sexes below pubertal age

disease produced by the female *Acarus scabies* and is characterized by vesicles

Etiology Scabies is quite common in the tropics It forms about 15 p c of all skin diseases Age it affects all ages but is commonly seen in children Intimate contact with a patient causes infection Common in winter

Sex—Both sexes are equally affected

The Acarus scabii which is also known as Sarcoptes scabii is a minute living mite which is about 1/60th of an inch in size and in shape it is ovoid Is has four pairs of legs The female causes the disease called scabies It lays its eggs in the skin for which it burrows in the epidermis and as it goes deeper the eggs are left behind The eggs hatch and larvae come out in about 3 days and move on the skin surface The larvae moult into nymph twice and then moult into the male and female in 17 days time After copulation the male dies and the female digs her own grave and she goes deeper in the skin About 50 eggs are laid The female dies after about 6 weeks The burrows are in length about 1/8th to 1/4th inch Common in winter and during war, femine and the like

Signs and symptoms The lesions are vesicular and sometimes erythemato papular The burrows can be made out with a hand lens on the front of the wrist It is very itchy particularly when in bed The distribution is typical the vesicles may by found along the anterior axillary folds the areola the umbilicus the linea alba, the inguinal ligaments, the genitalia and the perineum Round the shoulders round the elbows, front of the wrists and webs of the fingers (Fig No 131) Round

TRICHONOCARDIOSIS AXILLARIS

Definition Is a kind of fungus infection of the hair of axilla making it lusterless

Etiology Is due to the infection of the hair with Nocardia tenuis Varieties are (a) Black, (b) Yellow, (c) Red Red and black type usually occur with chromogenic cocci

Signs and Symptoms Only the cortex of the hair is infected and the hair becomes lusterless in the axilla

Diagnosis (1) Lusterless hair of axilla, (2) Examine the hair with 10 p c Sodium hydroxide

Defferential diagnosis Ringworm

Prognosis Good

Treatment Shave the hair and apply Ung Whitefield and hygiene is to be followed

DERMATOZOONOSES

Zoonosis means the dwelling of living insect in the skin such as scabies, microfilaria ankylostoma, dracontiasis etc Epizoonosis means the dwelling of the living insect on the skin and on the appendages of the skin such as pediculosis where the louse lives on the skin and on the hairs

Common zoonoses are (1) Scabies, (2) Filariasis cutis, (3) Dracontiasis cutis, (4) Creeping eruption, (5) Myiasis, (6) Ground itch, (7) Amaebic cutis, (8) Oxyuriasis dermatitis and (9) Dermal leishmaniasis

SCABIES

Definition Scabies is a contagious and itchy skin

Treatment Prophylaxis consists of daily bath and washing of clothings regularly which is done as a routine in the tropics hence the incidence of scabies in normaltime is neglible Curative consists of (1) Hygiene—this consists of sterilizing of all linens used by the patients by boiling and the woolen garments by ironing (2) Medicinal—the patients after bath with good scrubbing with soap applies the following ointment for a week and changes his clothings daily

Sulphur ppt gr 25
Hydrarg Ammon gr 10
Vaseline Alba oz 1

Ung for external use all over except face

If the patient follows the hygiene well and rubs this ointment every day after bath he gets well in 5 to 7 days Some patients may return after a week with itching and with erythematous le ions all over the body This may be either reinfection or usually sulphur dermatitis If the distribution is not typical of scabies the patient is treated with liniment Calamine for 2 to 5 days

If a quicker result is aimed at, instead of the above ointment the patient after soap water bath and scrubbing is painted thoroughly from neck to the toe with 25 p c Benzyl benzoit emulsion which is allowed to dry up before putting on clothing In moderate infection one painting is sufficient but in severe infection the patient is painted again next day without bath On the third day the patient takes a bath and puts on clean clothing When the patient complains of pruritus after a week it is generally due to Benzyl benzoit dermatitis or reinfection

FILARIASIS CUTIS

Definition Filariasis cutis is caused by microfilaria



Fig No 13
Scabies on hands

the gluteal regions, medial side of thighs, round the knees, round the ankles and webs of the toes In non ambulatory patients and young children also on the palms and soles and in infants may be on the face and scalp Mild constitutional symptoms may develop in scabies infection Albuminuria may also develop

Diagnosis (1) Typical lesions which are erythemato vesicular, (2) Distribution is typical, (3) Demonstration of a burrow and finding out of a living acarus, (4) Biopsy histopathology shows a bulla on the superficial layer of stratum corneum and a tunnel in the stratum corneum going obliquely down to the stratum granulosum with eggs and the mite and another bulla at the bottom of the tunnel The two intra epidermal bullae with the tunnel give the apearance like a dumb bell In the dermis there is perivascular infiltration and slight edema

Differential diagnosis (1) Impetigo, (2) Urticaria, (3) Dermatitis herpetiformis, (4) Drug rash, (5) Bullous syphiloderma, (6) Erythema multiforme

Prognosis Good

Differential diagnosis (1) Lichen Planus, (2) Cellulitis

Prognosis Good In ochocerciasis it is grave only when the microfilaria migrates into the orbit

Treatment Prophylaxis consists of using mosquito nets at night and freeing the neighbourhood of mosquitoes and flies Curative consists of using Hetrazen tablet or Benocide tablet by mouth in the following dose one tablet thrice daily for 3 to 6 weeks Surgical removal of the onchocerca nodule and excision of loa loa are done

DRACONTIASIS CUTIS

Definition Is the skin disease caused by the invsion of the hypodermis with the guinea worm

Etiology It is quite common in the tropics The female guinea worm is responsible for the disease The guinea warm embryo is poured ~~form~~ from an infected person s foot and is ejected into the water when the patient gets down near a tank The embryo tries to find out a host which is a cyclops In the body of the cyclops it changes its shape and develops a tripartite tail This cyclops when gets into the stomach of a man with drinking water gets digested leaving the guinea worm larvæ in the stomach to develop and migrate to such parts of the body and comes out when in contact with water After the embryos are set free they pass through the stomach wall and copulate and the male dies The female guinea worm takes about a year and half to develop to its proper size of about 25 inches in length and in thickness is like that of a thin earthworm The female guinea worm gradually migrates to the skin with her head forward and can be seen and felt under the skin of legs or back

of different species resulting in dermatitis, lymphedema, nodular swellings, transitory and permanent swellings

Etiology Filariasis cutis is caused by *Wuchereria bancrofti* and *malayi* and by *onchocerca volvulus* and by *loa loa* The microfilaria is transmitted by mosquitoes Onchocerceiasis is due to Onchocerca volvulus which is transmitted by black fly (Simulieum) whereas loa loa is transmitted by mangrove fly

Signs and symptoms The incubation period is very long and is several months Lymphangitis with erysepelatous skin lesions are the earliest signs Periodically these conditions recur Sometimes lymphedema alone is present Pain in the axillae, groins, and scrotum may be associated with fever Later on elephantiasis of the extremities, scrotum, vulva and breast may develop There may be orchitis and hydrocele also On the elephantoid skin there may appear oozing, cracks, fissures and ulcers

In onchocerciasis subcutaneous nodules are also found on the scalp instead of elephantiasis In loasis transient tumours develop, of the size of a bettle nut, anywhere on the body and are known as *Calaber swelling*

Diagnosis (1) Examination of blood for Microfilaria and loa loa, (2) Skin scraping for onchocerciasis, (3) Blood picture shows eosinophilia and leucocytosis, (4) Biopsy—histopathology shows epitheloid cells and presence of giant cells may be found in the dermis Destruction of elastic fibers in the dermis with disappearance of the skin appendages may be found The microfilaria may be found in the dermis also

CREEPING ERUPTION

Definition Is a skin disease caused by the migration of the larva of the ankylostoma the gnathostoma or by the larva of tropical warble fly (Hypoderma)

Etiology Commonly the larvae of Ankylostoma braziliense is responsibe Larvae of gnathostoma are also responssible They all get attached to the skin and burrow deep After reaching the hypodermis they migrate leaving a serpigenous ulcer Both sexes and all ages are affected in the tropics

Signs and symptoms Erythematous nodules are commonly found on the back and buttocks Gradually the *nodules* disappear and superficial ulcer with an erythematous border is left which gradually spreads circumferentially and is called the *Creeping eruption*

Diagnosis (1) Typical serpigenous lesion, (2) Site back or buttocks (3) Blood picture eosinophilia, (4) Biopsy histopathology shows atrophy of the *epidermis* above the parasite and the body *of the larva*

Differential diagnosis (1) Scabies, (2) Myiasis

Prognosis Good

Treatment Prophylactic treatment is to clean the neighbourhood of jungles and logged areas and regular D D T spraying by the Public Health authorities Putting on proper clothing to avoid exposing the bare body

Curative consists of (1) Painting the part twice daily with a skin antiseptic (2) *applying twice daily* on the lesion a paint containing

Acid Salicylic	gr 15
Colloion flexile	oz 1

Signs and symptoms Found in adults of both sexes A bulla appears near the ankle which bursts after a day or two The ulcer is at the opening of the uterus Sometimes the uterus is prolapsed through the ulcer From the site of the ulcer a zigzag serpigenous papular elevation may be felt upwards which is the coiled female guinea worm There may be associated lymhangitis and urticaria

Diagnosis (1) Typical site of the *bulla or* ulcer and the guinea worm can be palpated from the site of lesion upwards to a great length, (2) lymphagitis, (3) when the leg is put in a bowl of *cold water the ejection* of whitish fluid from the ulcer can be observed, (4) Blood picture will show eosinophilia and leucocytosis, (5) Microscopic examination of a *drop of ejected fluid* will show guinea worm larvæ, (6) Biopsy—histopathology of the skin will show the body and uterus of the guinea worm

Differential diagnosis (1) Chronic sinus due to osteomyelitis, (2) Creeping eruption

Prognosis Good

Treatment Prophylaxis consits of (a) boiling the drinking water, (b) Stop *infected into* get down in the tank Curative (a) Before the bulla has formed injection of aquous mercuric chloride solution (1 in 1000) is done at several *places on the body* of the guinea worm to kill it which gradually gets absorbed in the body, (b) Excision and taking out of the worm, (c) By dipping *the foot with the* ulcer in a bowl when the uterus is prolapsed and a stick *is passed through* it and slowly the worm is pulled out

of forceps after dropping chloroform or ether on the sore and then the wound is dressed

GROUND ITCH

Definition Is an itching skin disease confined only to the feet and legs and is caused by the larva of ankylo toma in the tropics

Signs and symtoms It is due to the larva of anky lostoma duodenale The larva in penetrating the skin produces minute scratches which are very pruritic in nature Sometimes peticheal haemorrhagic lesions also develop Vesicular lesions are also seen Thus a local erythemato vesicular rash is produced which is very itchy and is called "*gro ind itch*" The inguinal glands often get enlarged due to secondary infection

Treatment Consists of prophylaxis use of foot wear Locally lotio calamine with or without Lotio Phenol (1 in 100) may be used Liniment calamine with 1 p c Liq picis carb detergens may be applied An ointment can be applied consisting of

Calamine ppt	dr 1
Hydrarg Ammon	gr 10
Acid salicylic	gr 10
Liq picis carb det	m 10
Vaselin Alba	oz 1

Ung for external use and keep the part bandaged Internally carbon tetrachloride and oil chenopodium are given in a mixture form followed by a saline purgative Cristoid (S & Z) is helpful

AMAEBIC CUTIS

Amoebic cutis is the most important and fairly com mon type of protozoal skin disease found in the tropics

(3) Antimony by parenteral injection is helpful such as Urea Stibemine (Brahmachari) intravenously starting with the smallest dose weekly and giving a total of 2 0 gram or Stibinol '100' (Stibanate) injecting intramuscularly biweekly starting with 0 5 cc and increasing by 0 2 c c in every injection until 2 0 c c per injection is given which is repeated 10 times

MYIASIS

Definition Myiasis is a skin disease produced by the larva of a fly

Etiology -The fly may deposit its ova in the wound on the skin where the larvae is hatched out producing myiasis Besides this, larvae of different flies attacks the human skin and complete their full development

Signs symptoms Tender nodule of the size of a pea may develop which changes its place underneath the skin This nodule may become red, suppurate discharge pus and ultimately the fully matured fly

Diagnosis (1) Typical shifting nodular growths on the exposed parts of the body or the presence of maggots in a wound, (2) Blood picture shows eosinophilia, (3) Biopsy—histopathology shows either a larva or a developed fly covered over by atrophied epidermis

Prognosis Good

Treatment Prophylaxis is to keep the patient with multiple skin lesions under a mosquito net to avoid laying of eggs in sores by the flies or to use well covered garments so that the larvae of flies may not attack the skin Curative maggots are removed with a pair

PEDICULOSIS

Definition Is a skin disease caused by lice and is characterised by itching and impetigenous lesions

Etiology Is found in the tropics Due to better hygienic habits it is a less common disease in the tropics than in temperate climates Scarcely a case is found in the skin out-patient of a big teaching hospital in India for demonstration to the medical students

The louse is a blood sucking parasite on man Common varieties of louse infecting the human host are —(1) Pediculosis capitis affecting the scalp, (2) Pediculosis corporis affecting the body, (3) Pediculosis pubis affecting the pubic region

The colour of the louse changes according to the colour of the hairs to camouflage itself The colour may be from pale brown to blackish brown In shape also there is some difference The head louse is longer than the louse affecting the pubic region In size the louse varies from 1 to 4 mm in length The female louse is larger in size than the male louse It has three pairs of legs armed with hooks The eggs are deposited at the roots of hairs and are fixed with a color round the hair root There may be many eggs attached to one single hair Eggs are greyish white in colour and oval in shape Each louse produces about 300 eggs but about 50 p c do not hatch Affects all ages and both sexes

Signs and symptoms The louse may be found on the scalp, on the eye lashes and all over the body and over the pubic region Itching is the only symptom Itching produces scratching when coccal infection takes place

Etiology Entamoeba histolytica is the cause of the disease The cutaneous amaebiasis follows the (a) intestinal infection, (b) perineal abscess, (c) drainage of a liver abscess, (c) following the drainge of a ruptured appendix

The cutaneous amaebiasis is always secondary to intestinal amaebiasis

Signs and symptoms Chronic granulomatous ulcer develop round the anus, buttocks, perineum or around drainage of a liver absecess There may be slight dis charge on the granulation tissue History of amaebiasis

Diagnosis (1) Examination of stool for amaebiasis, (2) Typical sites of the granulomatous ulcers, (3) Histo pathology will show the presence of granulomatous change in the dermis and the presence of a vegetative form of Entamaeba histolytica in the dermis

Differential diagnosis (1) Cutaneous gumma, (2) Dermal leishmaniasis, (3) Lupus vulgaris

Prognosis Good

Treatment Prophylaxis is to treat with emetine any case of liver abscess before doing drainage

Curative is to inject daily emetine hydrochlor gr 1 intramuscularly for 9 days Together with the injections the patient is given by mouth Aureomycin (250 mg) capsule daily, one every 6 hours with Vitamin B complex, for 9 days The cutaneous lesions are dressed with 1 p c aquous solution of emetine hydrochlor or 3 p c Aureomycin ointment

Dermal Leishmaniasis is in next chapter

causes pruritus ani and vulvae as well as lichenified papular itchy dermatitis round the anus or around the anus and vulva in children

Etiology, Signs and symptoms The female thread worm deposits her eggs on the anal muco cutaneous junction When the egg is deposited and when the larvae are hatched out it produces intense itching of the anus Due to the scratching the skin round the anus and perineum become excoriated and thickened as well as brownish red in colour During scratching ova gets into the nails and the child when puts its fingers in the mouth reinfection takes place Insomnia develops

Diagnosis (1) Pruritus in the anal region of a child (2) Excoriation and lichenified brownish red lesion around the anus or around the anovulval regions in a child, (3) scraping from the anal region when examined under the microscope boat shaped enterobious eggs can be seen

Differential diagnosis (1) Condylomata lata (2) Contact dermatitis (3) Piles

Prognosis Good

Treatment (1) Gentian violet in cachet in dose of 2 5 mgm each, one such thrice daily for a child for a week is helpful (2) Cristoid (Sharp and Dhome) child dose followed by a saline purgative May be repeated after a month, (3) Nails of the child should be regularly clipped off and 1 p c Hydrarg Oxidi flavum ointment is applied to the fingers at night The child should be in bed pyjama at night

and impetigenised lesions develop There may be petichial haemorrhages also Lymphadenitis may be found Insomnia may develop

Diagnosis (1) Itching, (2) Demonstration of the louse or the nit (egg of louse), (3) Impetigo, (4) Microscopic examination of the hair with a nit or the louse

Differential diagnosis (1) Impetigo, (2) Eczema

Prognosis Is good Is grave when there is an incidence of a case of typhus or relapsing fever or trench fever near about

Treatment Prophylaxis consists of cleanliness and regular bath In an endemic region or when there is a case of pediculosis in the family it is advisible to (1) Dusting of all clothing with 1 p c DDT powder or 5 p c pyrethrum extract

Curative is done by using ordinary powder such as Pearl powder (Bengal Chemical) with 5 p c DDT for dusting of beds and clothings every day and the patient should take bath with a bland soap followed by application of 4 p c Ung Hydrarg Ammon for the whole body but for the scalp and eye lashes 1 p c Ung Hydrarg Oxidi flavum is advocated Sometimes Lethane hair oil for the scalp is useful 1 p c Gammexane in alcoholic solution can also be used after shampooing of the scalp One application may be sufficient Lorexane concentration (Imperial Chem Pharm) is such a preparation *Aerosol bomb is advocated for body louse* infection 25 p c Benzyl benzoit emulsion painting is used for the infection of body and public regions

OXYURIASIS DERMATITIS

Definition Oxyuris vermicularis (thread worm)

Varieties commonly seen are (1) Hypopigmented lesion, (2) Erythematous lesion, (3) Nodular lesion and (4) Xanthmatous lesion

Signs and symptoms Hypopigmented lesion of dermal leishmaniasis may occur on any part of the body but are usually seen on the face, around neck, back, chest, (Fig No 132) arms, forearms, hands and thighs Pin point macules appear which become larger in size and coalesce to form big sheets Hypopigmented patches (Fig No 133) are macular are non itchy and non scaly Few months



Fig No 132
Dermal Leishmaniasis
(Hypopigmented Stage)

after the appearance of hypopigmented patches butterfly erythema appears on the face The wings of the butter fly are represented by the erythematous lesions on malor regions and the body of the butterfly is on the nose This stage may persist for some months Butterfly erythema becomes prominent after the patient has

CHAPTER—XI

GRANULOMAS OF THE SKIN

Common granulomas in the tropics are

(1) Dermal Leishmaniasis, (2) Syphilis, (3) Yaws, (4) Pinta, (5) Tuberculosis, (6) Leprosy, (7) Granuloma anulare (8) Foreign body granuloma and (9) Granuloma inguinale

DERMAL LEISHMANIASIS

Two types are common in the tropics and particularly in India The type which is common in Bengal, Bihar, Orissa and Assam is popularly known as '*Brahmachari's Disease*' or Post Kala-azar Dermal Leishmaniasis The other type which is seen in other parts of India with a dry climate is known as "*Delhi Boil*" or Oriental Sore Distribution of Kala azar and oriental sore are not seen together These diseases are transmitted by sand fly called *Phlebotomus argentipes* The causative organism is a Leishmania

Dermal Leishmaniasis This disease is a sequelae to Kala azar and about 2 p c of Kala azar patients develop post kala azar dermal leishmaniasis Commonly found in lower India, Eastern Pakistan, Ceylone, Burma, Shyam and China The organism responsible is called Leishmania donovoni and the transmitting sandfly is known as Phlebotomus argentipes Dermal leishmaniasis occurs as a host parasite adjustment Dermal leishmaniasis usually occurs one to two years after Kala azar It is not related to oriental sore

in the dermis In the erythematous stage there is oedema in the dermis with dilatation of papillary vessels



Fig No 134
Dermal Leishmaniasis
(Nodular stage)

with perivascular infiltration Parasites can be found in this stage in the dermis In the nodular stage there is thining of the epidermis with flattening of the papillae There is not marked change in the papillary layer of the dermis but the reticular layer of the dermis shows dilatation of vessels with perivascular macrophage proliferation Large number of parasites are found in these macrophages In the xanthomatous stage no L D body is seen (6) Complement fixation test using Leishmania bodies as an antigen is found positive quite early and can even be used as a check to prognosis and follow up Patient should not be considered cured until this test is negative

Differential diagnosis (1) Lupus Erythematosus (2) Lupus Vulgaris (3) Leprosy, (4) Leucoderma (5) Pellagra and (6) Xanthoma Tuberosum

Treatment Antimony is the only treatment

exposed himself in the sun for sometimes This erythematous stage is followed by nodular stage on face first (Fig No 134) and then on other parts of the body



Fig No 133
Dermal Leishmaniasis
(Hypopigmented Spotage)

Nodules are deep brownish in colour and are soft to the feel The stage persists for many years and may or may not finally develop into the Xanthomatous stage Xanthomatous stage is very rare In the xanthomatous stage the patient develops yellowish plaques which resemble xanthoma tuberosum

Diagnosis (1) History of long continued fever or having had treatment for Kala azar about a year or two back, (2) Aldehyde test is negative, (3) Antimony test is negative, (4) Skin snip smear is positive for L-D bodies when stained with Leishman stain, (5) Biopsy is stained with Hæmatoxylin and Eosin and the histopathology shows in the hypopigmented stage decrease of melanin in the stratum basalis with dilatation of papillary vessels, Parasite may or may not be seen

Diagnosis (1) Smear from lesion shows leishmania tropica, (2) Culture of smear shows leishmania tropica

Differential diagnosis (1) Gummatous ulcer, (2) Desert sore (3) Tuberculous ulcer, (4) Diphtheretic ulcer

Treatment Dressing with an antiseptic is usually done Base of the ulcer may be infiltrated with 1% Atebrin solution or 1% Berberine sulphate Dressings with an ointment containing Zinc oxide dr 1/2, Acid salicylic-gr 10 Hydrarg Ammon gr 10, Vaseline Alba oz 1



Fig No 135
Oriental Sore
(Case of Dr S C Desai)

SYPHILODERMA

Syphiloderma is the commonest manifestation of syphilis and is caused by Treponema pallidum

In the acquired cases syphiloderma develops about 2 weeks after infection and is called "Hard Chancre Genital and there may also be extra genital hard chancres on lips, fingers, and anal margin

Urea stibamin (Brahmachari Research Lab Calcutta) starting with 0 01 gram in 2 c c pyrogen free distilled water is injected intravenously after examining the urine for the presence of albumin and also blood for agranulocytosis Total dose should be 2 5 gram per course Injections may be given twice weekly with high protein diet and citrous fruits This course is to be repeated 6 to 10 times with intervals of 1 month Other Antimony preparations may be used both I M and I V Stibanate (Gluconate) 2 c c inj I M biweekly Total dose 30 c c repeating 5 to 6 times with one month's interval

Toxicity of antimony—vomiting, giddiness, cold clammy skin and even signs of collapse Pulse becomes rapid with headache, discomfort in chest, joint pains and even jaundice Adrenalin hydrochlor (1 in 1,000) should be injected I M immediately with the development of any signs of toxicity and thereafter 500 mg Vitamin C intramuscularly or with I V 25% Glucose 25 c c Liver extract (crude) I M inj biweekly 12 such

Oriental Sore Known as Delhi boil or tropical sore *Found commonly in upper India and Western Pakistan* and it is not a sequelae of viseral Kala azar Oriental Sore is found on the exposed parts of the body and face may be first affected (Fig No 135) with induration and a dirty base In size varies from ½" to 1' diameter and is almost circular in shape Starts as a papule on which scales appear Crusting takes place soon and the ulcer breaks down and slowly extends peripherally Ulcers heal with a slightly depigmented depressed scar The sore is inoculable and usually gives protection against reinfection

Gummatous syphiloderma occurs anywhere on the body (Fig 136) but are commonly seen on the face (Fig No 137) lower legs, trunk, arms and scalp



Fig No 136
Syphilitic cutis
(Multiple gummatus ulcers)



Fig No 137
Congenital cutaneous syphilis
(Showing destruction of the bridge of the nose and the upper lip)

Early secondary syphiloderma develops about 2 months after the infection and is characterized by rashes all over the body with glandular enlargement There is bilateral enlargement of the cervical axillary, epitrophlear and inguinal glands which present a painless, nodular and indiarubbery feel Rashes are pleomorphic in type that is a mixture of various kinds as follows (a) Macular-this may again be (i) Roseolar and (ii) Pigmentary in nature (b) Papular this may be subdivided into (i) Squamous, (ii) Erythemato squamous, (iii) Vesicular, (iv) Hypertrophic, (v) Ulcerative, (vi) Mucous patches such as Condylomata lata

Important characteristic of rashes are (1) Distribution is symmetrical and bilateral, (2) Configuration tendency to get arranged in circles, (3) Colour primary rashes are pinkish but secondary rashes are brownish red, (4) Induration present in the early secondary rash and is absent in early primary rashes

Late syphilodermic rashes are as follows

(1) Pigmentary syphiloderma-macular lesion is common on palms and soles of white and black in colour May rarely occur over the neck called "collar of Venus" Atrophic changes may occur, (2) Nodular syphiloderma-nodules are circular, central necrosis may appear giving rise to a 'punched out" ulcer or the nodules may remain so for years, (3) Squamous syphiloderma is seen commonly on palms and soles This is true nodular syphilide but produces scaling and is dull red, (4) Gummatous syphiloderma gradually becomes larger in size Central softening occurs and gets adherent to the skin above The skin breaks down and forms a "punched out' ulcer with a "wash leather" base

changes of fingers and toes, (6) Syphilitic wig—is the excessive growth of hair in congenital syphilis Syphilitic wig may be followed by patchy alopecia called 'moth eaten" alopecia, (7) Generalised adenitis is commonly seen in congenital syphilitics (8) Dactylitis, osteochondritis, craniotabes, Parrot s nodules and "hot cross bun" appearance of the skull are commonly seen

Diagnosis (1) Dark ground examination of smear from the ulcer for Treponema pallidum, (2) Fontana's stain for T pallidum, (3) Wassermann Reaction, Blood W R is pesudo possitivė in (i) Leprosy (ii) Psoriasis, (iii) Chronic malaria, (iv) Infective mononucleosis (v) Diabetes and (vi) Physiological condition like pregnancy and (vii) Cancer Hence it is advisable to do (4) Kahn Test of blood, (5) Histopathology shows infiltration with plasma cells in the dermis with endothelial swelling

Differential diagnosis (1) Hard chancre should be differentiated from (a) Traumatic ulcer, (b) Chancroidal ulcer, (c) Scabies, (d) Herpes progenitalis, (2) Roseolar syphiloderma from (a) Measles, (b) Drug Rash (c) Pityriasis rosea, (3) Pigmentary Syphiloderma from vitiligo, (4) Squamous syphiloderma from (a) Ring worm, (b) Tuberculoid leprosy (5) Erythemato squamous syphiloderma from (a) Psoriasis (b) Seborrhoea (6) Vesicular syphiloderma from (a) Scabies (b) Impetigo, (c) Pustular acne vulgaris (7) Hypertrophic syphiloderma from (a) Yaws and (b) Pemphigus vegetans, (8) Ulcerative syphiloderma from Ecthyma, (9) Condylomata lata from condylomata accuminata and papilloma

Treatment Should be preceeded by the (1) Examination of Urine, (2) Examination of Blood W R

Congenital Syphiloderma is characterized by macular, papulo squamous or bullous lesions on the palms, soles, buttocks and on face Associated with the skin manifestations there may be (1) "Saddle nose", (2) Laryngitis (3) Rhagades deep fissuring in the line of the normal skin folds which on healing leave linear fissures, (4) Circum oral nodulo c utaneous is commonly seen (Fig No 138)



Fig No 138
Congenital syphilitic cutis
(Annular syphilide in
a girl aged 8 yeers)

Commonly on the ano genital (Fig No 139) muco cutaneous junctions, angles of the mouth (5) Onychia nail

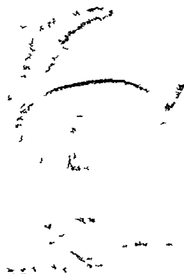


Fig No 139
Congenital syphilitic cutis
with condylomata lata anum
(Boy aged 3 years)

patient may be declared cured Repeat courses at intervals of one month

Arsenicals commonly used are Acetylarson, N A B, and Thiosermine (Brahmachari Research Lab Calcutta)

Course consists of weekly intramuscular injection in empty stomach of Thiosermine 0 15 gram one injection, 0 30 gram one injection 0 45 gram one injection and 0 60 gram seven injections Total 5 0 gram per course Urine is to be examined for albumin before every injection

Bismuth preparations commonly used are colloidal Bismuth metal in O 2 gram intramuscular weekly injections for 10 weeks making a total of 2 O gram Mercury is used with Iodide in mixture Liquor Hydrarg Perchlor m 15, Potassium Idide-gr 5 Tr Cinchonaco m 6, Aqua oz 1, Ft mist for a dose after food thriced aily

Adjuvant It is always beneficial to give the patient high protein diet with plenty of green vegetables and oranges to protect the liver as a prophylaxis against post arsenical intoxications

For Leprosy and Tuberculosis see Chapter VIII

YAWS

It is also called *Frambesia tropica*

Definition It is a chronic non venereal skin disease characterized by various skin lesions caused by Treponema pertenue

Etilogy It is found all over the tropics In India it is only seen in the States of Assam It is caused

(3) Examination of Blood Kahn, (4) Examination of Blood for total counts of R B C and W B C differential count of W B C and Haemoglobin p c, (5) When possible blood is examined for Vitamin C estimation of fasting blood It has been observed that when the blood Vitamin C is lower than 0 4 mg per 100 ml of blood the patient is likely to develop post arsenical complications during arsenic therapy

The treatment should be the combined Penicillin Arsenic Bismuth Mercury therapy

Penicillin should be given for 10 days by the injection of aquous crystalline penicillin "G" 2 5 lacs twice daily at 12 hours interval with no other treatment or 0 5 Mega unit is injected intramuscularly once every day In 10 days the total penicillin dosage is 5 Mega unit (1 Mega unit is 10 lacs unit of penicillin) Antihistaminics and Vitamin C togeher orally or parenterally may be used in penicillin drug rash which may occur as a complication to the antibiotic therapy

From the 11th day of treatment the following course is given Arsenicals by injection weekly for 10 weeks, Bismuth by injection weekly for 10 weeks Mercury and Pot Iodide by mouth for 14 weeks in form of a mixture

Followed after a month by the examination of (a) Blood W R and (b) Blood Kahn Tests

If tests are negative repeat 3 courses of Arsenic Bismuth-Mercury and follow up the case for 2 years more with blood and clinical examination 4 times a year When everything is negative at the end of 2 years after completion of the anti syphilitic therapy the

tartiary stage ulceration of the skin called "gumma" and sometimes circinate type of squamous lesions may be seen Depigmented or hyperpigmented patches Juxta articular nodules goudou and mutilation of rose and nasopharynx called *Gangosa*' may be seen

Diagnosis (1) Typical lesion on the skin, (2) Treponema pertenue on dark field examination, (3) Blood W R pseudo positive (4) Biopsy histopathology shows acanthosis of stratum mucosum where trepo nema pertinue may be found Infiltration with round cells and plasma cells in the dermis

Differential diagnosis (1) syphilis, (2) Ringworm, (3) Scabies (4) Lichen planus (5) Seborrhoea, (6) Lupus vulgaris, (7) Leprosy

Prognosis Good except in very advanced stage

Treatment Prophylaxis is to avoid contact with an infected patient Curative Every ulcer must be covered (1) Acetylarsone (Stoversol) O 25 gram tablet thrice daily for a week by mouth, (2) Penicillin crystilline O 5 Mega unit in aquous solution is injected intra muscularly every day for 2 weeks Arsenic Bismuth injection Thiosermine (Brahmachari Calcutta) O 15 gram one O 30 gram one O 45 gram one, O 60 gram to be injected weekly intramuscularly after urine exami nation and Bismuth O 2 gram is injected intramuscularly once every week Open wounds should be dressed with 4 p c Hydrag Ammon ointment

PINTA

Definition Is a chronic non venereal skin disease caused by *Trepona carateum*

by **Treponema pertenue** It affects all ages and both sexes Existing ulcer in the skin is necessary for infection Incubation period is 3 to 6 weeks

Sings and symptoms It has three stages called (1) Primary stage, (2) Secondary stage and (3) Tartiary stage It starts in the primary stage as a single macular lesion on the skin called "*Yaws spot*" A single cauliflower like granulomatous lesion on the skin is called '*Mother Yaw*" The secondary stage comes 3 months after the primary stage Maculo papular or maculo squamous lesions which appear on the planter surface mainly and nodular growths (Fig No 140) on face

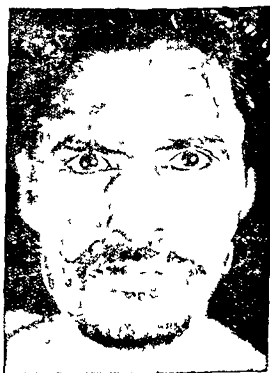


Fig No 140
Yaws
(Secondary Stage)
(Case of
Dr P Damodaram)

and body which is called the "*Crab yaw*" Sero-purulent discharge is present Joint pain is called "yaws pain" and itching are common symptoms at this stage he

Etiology Quite common in the tropics Cause is not known Both sexes and all ages are involved

Types (1) Ordinary type, (2) Giant type

Signs and symptoms Papular and skin coloured half pea sized lesions with a tendency to arrange in the form of a ring with a diameter of ¼ ½ to ¾ inches called ordinary type while rarely very large sized lesions are seen called giant type There is no itching and no pain Single lesion seldom occurs except in the gaint type but in the ordinary type the lesions are about half a dozen in number Site commonly found on the knuckles of fingers and are also seen over the back of the wrist round the knee round the gluteal regions and the ankles Rarely may be found at other places like scalp

Diagnosis (1) Ringed skin coloured lesion on the back, hand elbow, knee, ankle joints without any itching or pain, (2) Histopathology shows dege neration of the connective tissues and presence of epitheloid cells in the dermis

Differential diagnosis (1) Erythema multiforme, (2) Leprosy, (3) Syphilitic cutis, (4) Lupus vulgaris
Prognosis is fair rarely cured

Treatment Locally may be applied in an ointment containing

Acid Salicylic gr 10, Crude coal tar dr ½ Vaselin alba oz 1

X ray therapy is also advocated High dose of vitamin A is also useful When all other treatment proves useless multiple incisions on the ring down to the dermis is helpful

Etiology It is found in certain parts of tropics It is rare in Asia but has been reported from Africa Affects adults of both sexes Incubation period is a week

Signs and symptoms Two tages such as (1) Primary and (2) Secondary *In the primary stage* erythemato squamous lesions are seen Lymphadenitis may be present Secondary stage appears about one year after infection Erythemato squamous lesions are mainly found on the face and extremities On the plam and soles and also on the dorsum of feet and hands papular pigmented lesions alternating with vitilaginous macular lesions are seen which is known as the **late dyschromic stage**

Diagnosis (1) Vitilaginous macular lesions alternating with pigmented macular lesions on the hands and feet in an adult, (2) Blood W R positive, (3) Biopsy histopathology shows microabscesses and acanthosis with degeneration of the stratum basalis In the dermis there may be found *Treponema c rateum* with solid masses of chromrtophores

Differential Diagnosis (1) Leucoderma, (2) Syphilis
Prognosis Good

Treatment Penicillin crystallin 'G' aquous solution intramuscularly is injected O 5 Mega Unit daily for 15 days Arsenic bismuth may be used also

GRANULOMA ANULARE

Also known as ringed eruption or lichen annularis
Definition (Is a skin disease of unknown cause and is characterised by skin coloured ringed lesions



Fig No 141
Granuloma Inguinale
(Case of Dr B S Verma)

Diagnosis (1) Chronic granulomatous ulcer in the genitalia spreading either to the groins or to the perineum or both in adults (2) Skin test of Anderson positive, (3) Histopathology shows in the dermis dense small round cells, large histiocytic cells with Donovan bodies within them

Differential diagnosis (1) Cutaneous syphilis, (2) Chancroid, (3) Tropical Bubo from Psendo bubo

Prognosis Fair

Treatment (1) Antibiotic such as Penicillin injection 0 2 Mega unit twice daily by intramuscular injection for 2 weeks, (2) Aureomycin capsule (250 mg) every 6 hours for 2 weeks (3) Streptomycin gram 1 injected intramuscularly daily for two weeks, (4) Achromycin or Iloticin may be useful, (5) Locally 3 p c aureomycin ointment is advocated

FOREGN BODY GRANULOMA

Definition Is a granuloma due to the introduction of a foreign body in the dermis

Etiology Common forign body is the wax introduced into the skin of the left hand accidentaly by shoe makers in the tropics, thorn or a fish bone Painless signs and symptons nodular growths on the skin of hands or anywhere on the body No suppuration or erythema seen

Diagnosis (1) Painless, non suppurating indolent growths nodular (2) Histopathology shows the wax by special stain

Differenital diagonosis from other granulomas

Prognosis Good

Treatment Excision

GRNULOMA INGUANLE

Definition It is a chronic contagious skin disease characterized by ulcerating granuloma of the genitalia and groin

Etiology Sex—occurs in either sex Age—young adults are commonly affected in the tropics It is acontagious and is said to be of venereal origin Causative organism is an intracellular body called *Donovan body* This is a gram negative bipolar body

Signs and syptoms A granulomatous ulcer (Fig No 141) on the external genitalia which gradully spreads towards the groin above and towards the perineum below It takes the from of subcutaneous nodule called **pseudobobo** The spread is very slow

of different shapes and are macular (Fig No 142) Mongolian spot and supernummary ear and super numary finger are also birth marks



Fig No 142
Pigmented Nevus
or birth mark
(Covering infrascapular region and back of arm)

Sometimes the moles grow hairs on them and are called hairy naevus or **Naevus piolosus** It is said

Fig No 143
Giant Pigmented Nevus
Pilosus
(Bathing Trunk type)



CHAPTER XII

TUMORS OF THE SKIN

Tumuors of the skin are mainly of four different types (1) Benign Epidermal, (2) Benign Dermal, (3) Precancerous and (4) Cancer

Common benign epidermal tumours are (1) Naevus, (2) Milium, (3) Sebaceous cyst, (4) Chondrodermatitis Nodularis Chronica Helicis, (5) Adenoma Sebaceum, (6) Multiple Benign Cystic Epithelioma and (7) Cylindroma

Naevus—This is the common mole Every human being has moles There are always multiple moles on the body Some people have many moles others have just few here and there adding to the beauty when on the face With the growth of age the moles increase in number There may or may not be any mole at birth but may appear any time after birth

Hence naevus may be classified into (a) Birthmark and (b) Mole Moles are of various types such as (1) Pigmented mole, (2) Pigmented hairy mole, (3) Verrucous mole, (4) Giant mole, (5) Naevus linearis, (6) Junction naevus

Moles are about the size of a pin head or half pea-sized generally They are either macular or slightly papular Moles are usually completly black in colour Moles occur anywhere on the skin of the body Some times a mole is quite large in size and is called a "Birth mark" Birth marks are rectangular, oval or

Rarely the mole is unilateral and is confined to one side of the body when it is called **Naevus linearis** (Fig No 146) This is an unilateral mole and may be confined only to one of the extremities or it may run from the ankle and proceed upwards along the medial side of the leg and thigh and reach the ischial tuberosity and then up the gluteal region along the side of the body to the axilla of the same side and down the medial side of the arm and forearm ending at the front or back of the wrist or sometimes passing over the dorsum of the hand ending infront of one of the knuckles



Fig No 146
Naevus linearis

Junction naevus is the one which undergoes malignaney

Diagnosis (1) Pigmented mole is either macular papular verrucose, linear or hairy (2) Size may be pin head or half pea sized commonly but sometimes it is of a giant type when it covers either the whole of the trunk or parts of limbs or face (3) Does not undergo malignacy except those which are junction naevi, (4) Histopathology—Eqidermis is thin Below the eqidermis groups of naevus cells and sometimes giant cells are found Neavus cell is oval with a large nucleus In the verrucous type there is hyperkeratosis with acanthosis and elongation of the rete pegs together with groups of naevus cells

that naevus pilosus never goes malignant Sometimes they are very big and cover the whole of the trunk and are called bathing trunk type of neavus pilosum or giant type (Fig No 143) Not uncommonly verrucoid growths are found on a pigmented mole and is called **Naevus Verrucosum** (Fig No 144 & 145)



Fig No 144
Naevus Verrucosum



Fig No 145
Pigmented nevus

Differential diagnosis (1) Dermoid cyst and (2) Molluscum contageosum,

Prognosis Good

Treatment Surgical removal

CHONDRODERMATITIS NODULARIS CHRONICA HELICIS

It is a nodular painful condition of the helix of the ear Single or multiple nodules of the size of a pea on the helix are found Wrestlers and boxers get it due to the trauma on the ear but rarely occurs without trauma The nodule is attached to the cartilages of the ear When ulcerates it becomes persistent Never goes malignant Commonly seen in men

Diagnosis (1) Situated on the helix of the ear, (2) Painful lesion, (3) Biopsy—Acanthosis is present in the epidermis Dermis shows inflammation and infiltration with round cells and some giant cells There is degeneration of collagen and fibrous tissue also The underlying cartilage shows inflammation

Differential diagnosis (1) Epithelioma, (2) Senile keratosis and (3) Eczema

ADENOMA SEBACEUM

Found in both sexes Is often hereditory Disease starts in childhood Pin head to half-pen sized nodules appear on the face (Fig No 147 & 148) They are yellowish in colour but some are reddish when fine blood vessels are seen on the nodules Associated abnormalities may also be present such as warts, naevi, pigmented

Differential diagnosis (1) Warts, (2) Molluscum contagiosum, (3) Basal cell carcinoma, (4) Melanoma

Prognosis Is good when a growing naevus is excised

Treatment Moles occuring near the places of trauma should be removed by excision Plastic operation is advised for giant naevus When naevus is growing in size it should be removed Junction naevus should be removed

MILIUM

Skin coloured pin head sized lesions situated on the face particularly on the forehead and around the eyes Often seen in children but may occur at any age It is neither tender nor painful It never ulcerates and remains as it is for years

Histopathology shows a horny cyst with a thin epidermis The horny mass looks like onion

Differential diagnosis (1) Acne comidone, (2) Molluscum contagiosum

Pronosis good

Treatment Incising and scraping the horny cyst

SEBACEOUS CYST

Sebaceous secretion is poured into the pilo-sebaceous follicle The cyst is formed by the occlusion of a hair follicle It is an oval tumour and is soft and elastic Reacton is present Cyst is yellowish and semisolid in consistency Sometimes a cyst becomes infected May occur anywhere on the body Multiple sebaceous cysts occur on the scrotal skin which may undergo calcification.

Differential diagnosis (1) Dermoid cyst and (2) Molluscum contageosum,

Prognosis Good

Treatment Surgical removal

CHONDRODERMATITIS NODULARIS CHRONICA HELICIS

It is a nodular painful condition of the helix of the ear Single or multiple nodules of the size of a pea on the helix are found Wrestlers and boxers get it due to the trauma on the ear but rarely occurs without trauma The nodule is attached to the cartilages of the ear When ulcerates it becomes persistent Never goes malignant Commonly seen in men

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spots Sometimes there is a history of mental disease, epilepsy, heart disease or visceral growths May be familial

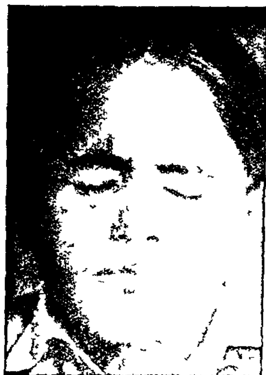


Fig No 147
Adenoma sebaceum
(Boy aged 7 years with history of epilepsy)

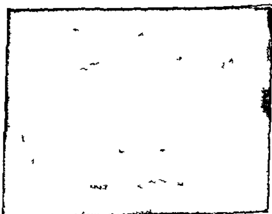


Fig No 148
Adenoma Sebaceum
(Girl aged 8 years)

Diagnosis (1) Early onset, (2) Nodules on face, (3) Histopathology shows hyperplasia of the hair follicles with hyperplasia of sebaceous glands and blood vessels

Differential diagnosis (1) Acne vulgaris, (2) Acne rosacea, (3) Colloid milium, (4) Molluscum contagiosum, (5) Multiple benign cystic epithelioma

Prognosis Is never cured

Treatment Removal of lesions is the treatment of choice X ray therapy is also helpful

MULTIPLE BENIGN CYSTIC EPITHELIOMA

Pin head to pea sized yellow nodules occur on face at pubertal age Lesions are symmetrically arranged

Diagnosis (1) Situated on the face, (2) Age of onset is at puberty, (3) Histopathology show proliferation of the cells of stratum basalis

Differential diagnosis Cylindroma

Prognosis Good

Treament Removal of the tumours surgically

CYLINDROMA

Commonly known as turban tumour Occurs o the scalp In size vary from a pea to an agg In number may be single or multiple and the colour is that of the skin Very slowly increases in size and rarely becomes a very big tumour looking like a turban on the head Sometimes there is a history of injury to the head There is often associated alopecia seen Often familial Turban tumours are derived from the coils of sweat gland

Diagnosis (1) Situated on the scalp, (2) Skin coloured tumour of the size of a pea to that of an

agg on head, (3) Histopathology- shows masses of basal cells and hyaline degeneration side by side

Differential diagnosis (1) Granuloma pyogenicum, (2) Sebaceous cyst

Prognosis If untreated may undergo malignancy

Treatment Removal by operation

COMMON BENIGN DERMAL TUMOURS

They are (1) Scar, (2) Keloid, (3) Fibroma, (4) Neurofibroma, (5) Neuroma, (6) Lipoma, (7) Glomus tumour, (9) Osteoma, (10) Lymphangioma and (11) Angioma

SCAR

Is also known as cicatrix Scar is the new formation of connective tissue in the skin as a result of a skin disease or trauma

Signs and symptoms Scar is at level with the in It is generally of the colour of the skin car may assume any shape It is smooth and shiny here is no hair on the scar Occurs at all ages id in both sexes

Diagnosis (1) Smooth, shiny, hairless patch of tissue on level with the skin, (2) Histopathology absence of hair follicle and sweat glands There are bundles of interlacing connective tissue with no blood vessels

Complication Scar may become hypertrophied and results in keloid formation

Prognosis Good

Treatment No treatment is necessary until complication develops

KELOID

Definition Is a tumour arising from the fibrous tissues of the dermis

Etiology Heredity has some influence Cause is not known Trauma is an exciting factor but sometimes chronic inflammation of the hair follicle is also responsible Some think that the keloid begins by hyperplasia of round cells in the adventitia of arterioles

Classification 2 types (1) Idiopathic and (2) Secondary Age—may occur at any age Sex—both sexes are equally affected

Signs and symptoms Starts as a nodule on the normal skin but usually on a scar (Fig No 149)

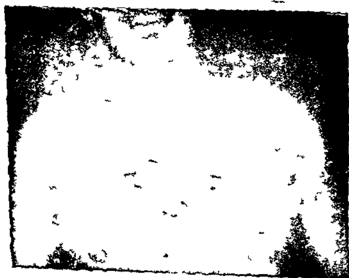


Fig No 149
Post acne Keloid

This nodule raises the skin above it and generally enlarges in size Sometimes becomes slightly red and tiny processes proceed from its base like the crow's claw (Fig No 150) It is hard to the feel



Fig No 150
Keloid

Diagnosis (1) Growth which is hard to the feel and of skin colour, (2) Claw-like processes at the base of the tumour, (3) Histopathology—Hyperplasia of the connective tissues of the dermis Cellular elements are present in a new keloid but are absent in an old one

Prognosis Good with treatment Some disappears spontaneously

Treatment Repeated injection of Hyaluronidase is helpful in small keloids But for keloid of very big size excision and radiotherapy The usual procedure for a moderate sized keloid is to give X-ray therapy in suberythema dose twice at weekly intervals foll ed by excision and after the wound heals two more su thema

doses of X ray exposure at weekly intervals Sometimes radium application is slso helpful

FIBROMA

Etiology Is not known

Commonly seen in elderly people at climacteric Found in both sexes

Signs and symptoms Single or multiple, pedunculated and soft growth of skin colour seen commonly on face, neck or other parts of the body In size like that of a pea

Diagnosis (1) Skin coloured single or multiple pedunculated soft growth and in size like a pea, (2) Histopathology shows fibrous tissue proliferation

Prognosis Good

Treatment No treatment is necessary Sometimes it is excised from the base of the peduncle and the base is cauterized

NEUROFIBROMA

Known as Von Recklinghausen's disease Is a condition characterized by the development of multiple tumours Skin develops a peculiar pigmented clour

Etiology Not known

Seen in both sexes but is commonly seen in males in the tropics Middle aged males are commonly seen with multiple neurofibromatosis and some are pedunculated Some think it to be due to some change in the germ plasm and it starts from intra uterine life

This nodule raises the skin above it and generally enlarges in size Sometimes becomes slightly red and tiny processes proceed from its base like the crow's claw (Fig No 150) It is hard to the feel

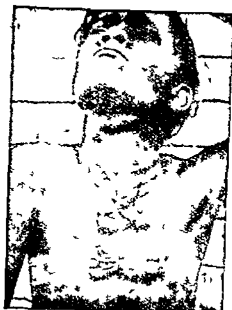


Fig No 150
Keloid

Diagnosis (1) Growth which is hard to the feel and of skin colour, (2) Claw like processes at the base of the tumour, (3) Histopathology—Hyperplasia of the connective tissues of the dermis Cellular elements are present in a new keloid but are absent in an old one

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fibrils with the absence of elastic fibres in the tumour With special nerve stain non medullated nerve fibres can be found



Fig No 152
Neurofibroma showing sesile tumors and cafe au lait coloured skin (Case of Dr K C Sahu)

Complication (1) Mucoid degeneration, (2) Malignant degeneration (sarcomatous change)

Differential diagnosis From (1) Leprosy (2) Mulluscum contagiosum (3) Multiple cutaneous sarcoma

Prognosis Bad Sarcomatous changes may occur Mental retardation has also been observed

Treatment Surgical removal when a single or a few or big

LIPOMA

Are multiple fatty tumours occuring in the dermis and in the subcutaneous tissue

Signs and symptoms Occurs all over the body from head to foot (Fig No 151)



Fig No 151
Neurofibromatosis

Tumours are soft to the feel In size tumours are pea-sized but gradually become bettle-nut in size and even larger weighing about a pound Skin shows (Fig No 152) one or two depigmented or hyperpigmented patches known as cafe au lait colour

Diagnosis (1) Multiple various sized tumours all over the body in a healthy individual, (2) Histopathology shows loose texture and wavy arrangement of the

Occurs on the extensor surface of the body and are symmetrical in arrangement Sites are face, neck, extremities and trunk, Seen commonly in young adults

Histopathology—shows smooth muscle fibers Muscle fiberes are found to originate from the erectori pilorum mu cle or from the muscular layer of blood vessel

Differential diagnosis From Syringocystoma

Prognosis Good

Treatment Freezing with carbon dioxide snow Excision of those which are pretty big in size

GLOMUS TUMOUR

Is also known as Angioneuro myoma

Signs and symptoms It is a rare tumour Small tumour which is soft to the feel and is painful It may be skin coloured or reddish Found under the nail Pain starts from the finger tip and radiates along the hand and the pain increases with cold There is no metastasis Lentil in size and found in both sexes

Diagnosis (1) Site under the nail of a finger, (2) Painful and pain increases when touched with a cold object, (3) Histopathology shows a network of nerves and arterioles It is an overgrowth of the glomus body There is dilatation of the vessels with hyperplasia of the surrounding cells

Differential diagnosis Neuroma, Melanoblastoma

Prognosis Good after excision

Treatment Excision of the tumour

Etiology, Not known Occurs in both sexes and at all ages

Signs and symptoms Soft tumours of varying sizes May be peasized or the size of a cocoanut May be single or multiple and is lobulated Common sites are neck and gluteal region Found also in the breast of females Grows slowly Skin coloured

Diagnosis (1) Slow growing skin coloured lobulated, soft tumour on the neck, gluteal regions, on the breast of a woman, (2) Histopathology shows groups of large fat cells bounded by connective tissue *and is encapsulated*

Differential diagnosis From Dercum's disease which is a painful fatty tumour

Prognosis *Good as it does not recur* after removal

Treatment excision

MYOMA

Is a muscle tumour arising commonly from erectoris pilorum

Etiology Not known Found commonly in women Can occur in any age

Classification Two types, (1) Dartoic myoma, (2) Leiomyoma Dartoic myomas are commoner than the Leiomyoma They occur on the penis and scrotum of males and on the labia majora and breast of females Grows slowly In size starts as a pea becoming an orange and may be pedunculated

Leiomyoma are hard nodular growths They are multiple and nonpedunculated Are pea sized usually

serpiginosum, (d) Naevus anaemecus (when blood vessels are absent), (e) Erethyma palmare hereditarium

(2) Cavernous angioma are (Fig No 153 & 154), (a) Superficial type (Strawberry), (b) Deep type



Fig No 153
Haemangioma Scalp
(Behind left ear)



Fig No 154
Haemangioma
Strawberry typ
(Case of
Dr R N Gupt

OSTEOMA

Is the deposition of bone in the skin and subcutaneous tissue Osteoma is a new growth This may be localized or generalized and progressive

Signs and symptoms May occur at any age and in both sexes As small seed like papules or in large plaques can occur on face or anywhere

Diagnosis (1) X-ray and (2) Histopathology typical bony tissue will be found

Prognosis Bad as it is progressive

Treatment Excision but is progressive to such an extent that excision becomes impossible

ANGIOMA

Is a vascular naevus

Signs and symtoms Occurs soon after birth or sometimes several weeks after birth Occurs as a red tumour which may be on level with the skin or may be elevated from the skin level and is deep May occur in any organ of the body except in cartilage May remain stationary or suddenly start increasing in size Often some disappear by the age of 2 years but others persist throughout life Occurs in both sexes It is painless Cavernous angiomas become larger when the child coughs or cries

Classification

(1) Capillary angioma are (a) Spider naevus, (b) Portwine stain (or naevus flammeus), (c) Angioma

(4) Angiokeratoma, (5) Telangiectasia are (a) Congenital telangiectasia (b) Hereditary haemorrhagic telangiectasia, (c) Generalised telangiectasia, (d) Naevus anaemicus

(6) Naevus Lymphangioma (Fig No 157)



Fig No 157
Naevuslymphangioma
(on the left scapular region of a boy aged 10 years)

Diagnosis (1) Colour of the lesion, (2) Painlessness, (3) Present since birth or some weeks after birth, (4) Histopathology shows dilatation of blood vessels in the dermis and proliferation of newly formed vessels In the portwine stain type the whole of the dermis is affected In the cavernous angioma there is dilatation of the capillaries and multiplication of the blood spaces which have endothelial lining Lymphagiomata shows cystic swellings in the upper part of the dermis Angiokeratoma shows great dilatation of blood vessels in the dermis which are filled with blood and with hypertrophy of the epidermis

(3) Mixed type such as capillary cavernous angioma and Salerosing haemangioma (Fig No 155 & 156)



Fig No 155
Sclerosing angloma
(On the front of right thigh)



Fig No 156
Histopathalogy of sclerosing haemangiom

(4) Angiokeratoma, (5) Telangiectasia are (a) Congenital telangiectasia, (b) Hereditary haemorrhagic telangiectasia, (c) Generalised telangiectasia, (d) Naevus anaemicus

(6) Naevus Lymphangioma (Fig No 157)



Fig No 157
Naevuslymphangioma
(on the left scapular region of a boy aged 10 years)

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Prognosis Good

Treatment Application of carbon dioxide snow as slush with acetone is helpful in most cases Repeated twice montly for a fairly long time Thorium-X application twice monthly for a long time Application of Radium is helpful in the elevated type Repeated when necessary after 3 to 6 months Cauterization by chemical or by electro cautery is helpful in the spider naevus and in telangiectasias Roentgenray therapy is helpful in the elevated types of angiomas Excision is helpful in lymhangioma and angiokeratoma

PRE-MLAIGNANT TUMOURS OF THE SKIN

Malignant cancers of the skin may be divided into two groups such as (1) Precancerous condition of the skin and (2) Cancerous condition of the skin

(1) Precancerous conditions of the skin There are certain skin diseases which ultimately become malignant and are called precancerous 20 p c of precancerous conditions in the tropics become malignant Pathologically some will show malignant changes but clinically do not present malignant picture Common precancerous skin lesions are —(a) Arsenical keratosis, (b) Senile keratosis, (c) Cornu cutaneum, (d) Leucoplakia, (e) Bowen's disease, (f) Erythroplasia of Queyrat

(a) Arsenical keratosis—This is the commonest precancerous skin disease as arsenic is used extensively as a therapeutic agent in medical practice It results from the ingestion of arsenic orally but rarely also

parenterally The skin change may be observed as early as within 30 days or after 30 years Arsenical pigmentation develops in about 50 p c cases in those undergoing arsenical therapy Arsenical keratosis occurs chiefly on the palm and sole as pin head to small pea like hyperkeratotic lesions When loosely adherent keratotic scales are removed and indurated superficial ulcer is seen This ulcer changes into a malignant lesion either as a fungating type or ulcerating type Sometimes multiple warty growths are seen all over the body Metastasis is rare

Diagnosis (1) History of ingestion of arsenic, (2) Keratotic growth or indolent ulcer on palm and sole or multiple warty growths on the body, (3) Histopathology shows squamous celled epithelioma Dyskeratosis with vacuolization of cells also occur

Differential diagnosis (1) Calosity, (2) Superficial epithelioma of the skin (3) Bowen s disease, (4) Minakhi

Prognosis Sometimes lesions disappear without treatment but may persist and undergo malignant change

Treatment Arsenic medication must be stopped Excision of the lesion should be done

(b) Senile keratosis Is characterised by papular pigmented plaques on the dorsum of the hands and face in old people which bleeds when dry crust is removed Lesions are multiple

Diagnosis (1) Old people of over 60 years age (2) Bleeding indolent ulcer or crust covered lesion on the dorsum of the hand or on the face, (3) Histoppthology shows hyperkeratosis, acanthosis,

thickening of the stratum granulosum Mitosis may be found in epidermal cells with squamous cell carcinomatous changes prominent

Differential diagnosis (1) Arsenical Keratosis, (2) Seborrhoic wart, (3) Xeroderma pigmentosa

(c) Cornu cutaneum Is a localized epithelial tumour Cutaneous horns develop from filiform papilloma, from a wart, from a naevus, from a sebaceous gland and from the mucous membrane

Cutaneous horn is usually a solitary lesion with an indurated base The growth is slow but rarely rapid *Common sites are the dorsum of the hands,* temples and ears Rarely it has been found also on the nose (Fig No 158), lips, penis and on the trunk The shape is like a horn-conical, twisted or of any irregular shape Seen in people over 40



Fig No 158
Cutaneous horn on nose

Diagnosis (1) Conical, twisted or irregularly hard lesion on the dorsum of the hands or on the face

usually with an indurated base, (2) Age of patient is above 40 years, (3) Histopathology shows hyperkeratosis and parakeratosis of the stratum corneum Squamous call carcinomatous changes are seen

Differential diagnosis Filiform wart or Senile keratosis

Prognosis Good when treated properly

Treatment Excision is the choice of treatment

(d) Leucoplakia Is characterized by whiteness and thickening of the mucous membrane of the tongue lip or vulva

Sharp marginated white pin head, coin sized or irregular areas with cracks on the tongue, lip or vulva Commonly seen in elderly people Leucoplakia of tongue and lips are common in men

Diagnosis (1) Advanced age (2) Typical irregular or circular sharply marginated white plaque on tongue, lips or on vulva (3) Histopathology shows hyperkeratosis, acanthosis with degeneration of the stratum basalis Inflammatory cells are present in dermis

Differential diagnosis (1) Thrush (2) Syphilitic leucoplakia, (3) Lichen planus (4) Lupus Erythematosus

Prognosis If diagnosed and treated *early* the prognosis is not bad When malignant changes have taken place the prognosis is grave

Treatment Vitamin A therapy in high dose (100 000 i u) daily is helpful even when *it has undergone* any malignant change Vitamin A therapy should be given a

long trial Locally 20 p c solution of Silver Nitrate to be painted When malignancy occurs excision or radio therapy should be tried

(e) Bowen's Disease Characterised by scaly single or multiple plaques on any part of the body Red, oozing and indolent lesion appears on removing the crust Can occur at any age and in both sexes

Diagnosis (1) Indolent scaly plaque on the body, (2) Histopathology shows hyperkeratosis, acanthosis with irregular downward proliferation on the epidermis with keratinization and vacuolization of some cells Clumping of nuclei with mitosis are seen Squamous cell epitheliomatous picture is presented



Fig No 169 Erythroplasia of Querrat of penis (Case of Drs C V Rajam P N Rangiah, P K Azeezullah and J S Serma)

Prognosis Good when diagnosed and treated early

Treatment, Carbon dioxide freezing is helpful in early stage Excision is the treatment of choice

(f) Erythroplasia of Queyrat Slow growing, indolent, red, lesion which occurs on the glans penis (Fig No 159) vulva and on the mucuous membrane of mouth Occurs in middle aged persons Regional lymph glands are not enlarged



Fig No 160
Histopathology of Erythreplasia of Queyrat
(Case of Drs R V Rajam P N Rangiah P R Azeezullah and J S Serma)

long trial Locally 20 p c solution of Silver Nitrate to be painted When malignancy occurs excision or radio therapy should be tried

(e) Bowen's Disease Characterised by scaly single or multiple plaques on any part of the body Red, oozing and indolent lesion appears on removing the crust Can occur at any age and in both sexes

Diagnosis (1) Indolent scaly plaque on the body, (2) Histopathology shows hyperkeratosis, acanthosis with irregular downward proliferation on the epidermis with keratinization and vacuolization of some cells Clumping of nuclei with mitosis are seen Squamous cell epitheliomatous picture is presented

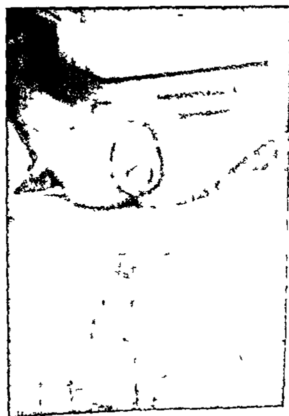


Fig No 159
Erythroplasia of Queyrat of penis
(Case of Drs C V Rajam P N Rangiah, P R Azeezullah and J S Serma)

Signs and symptoms Patients are sensitive to light and particularly to the ultra violet light Macular pigmentation on the exposed parts of the body appears



Fig No 161
Xeroderma Pigmentosum showing pigmentation and development of squamous cell epithelioma
(Case of Dr P N Behl)

Diagnosis (1) Indolent and red genital or buccal lesion in a middle aged person, (2) No response to any treatment, (3) Histopathology shows dyskeratotic changes with elongation of the rete pegs There is infiltration in the upper part of the dermis Squamous cell epitheliomatous changes gradually develops (Fig No 160)

Differential diagnosis (1) Chronic balanoposthitis, (2) Hard chancre, (3) Soft chancre, (4) Leucoplakia, (5) Leucoplakia of mucous membrane of mouth

Prognosis With early diagnosis and treatment it is good But when advanced the prognosis is grave

Treatment In the early stage in case of male genital circumcision and CO_2 application but in the late stage complete excision is advocated Complete excision is the treatment of choice also when affecting any other part of the body

MALIGNANT TUMOURS OF THE SKIN

Common cancerous conditions of the skin are

(a) Xeroderma Pigmentosa, (b) Melanoma, (c) Basal cell carcinoma, (d) Squamous cell carcinoma, (e) Reticulosis

(a) Xeroderma Pigmentosa is a skin disease characterized by pigmentation and development of malignant growths on the skin from the early age

Etiology found in the tropics On an average one case yearly attends a large skin *outdoor in India* Cause is not known It is an inherited skin disease Usually affects only one member in the family Both sexes are equally affected

In junction nævus the cell of origin is epidermal Junction nævus is an active nævus hence it is



Fig No 162
Melanoma Sole

potentially malignant The intraepidermal soft nævus may be hyperkeratotic papillomatous and verrucous in look Many contain hairs and hairy moles are benign Some are very large and are called giant moles

(2) Lentigo—is a very small deeply pigmented mark on the skin Histologically len igo is an early junction nævus and may develop into a melanocarcinoma

(3) Melanocarcinoma—arises from a junction nævus or a lentigo rarely from the normal skin Starts as a deeply pigmented rapidly growing tumour surrounded by a red hallow Later on the growth ulcerates and small pigmented lesion develop near it and becomes fungating Regional lymph glands are involved due to matastasis Lower extremity is the common site (Fig No 163)

Histologically large nævus cells with mitotic fingures and activity at the dermo epidermal junction are seen

(4) Blue nævus - occurs as an oval, flat blue

and increases each summer Patchy atrophy of the skin produces perchment skin Verrucous lesions develop on exposed parts Photophobia is a prominent feature In an advanced case the skin presents an exfoliative appearance, keratosis and malignant growths with telangiectasis (Fig No 161)

Diagnosis (1) Dry pigmented skin with photophobia and warty growths on face and hands, (2) Biopsy shows hypertrophy of the stratum corneum with atrophy of the stratum mucosum Hyperpigmentation of the stratum germinativum Verrucous lessions show acanthosis and closely packed pigmented cells Ulcerated lesions show basal cell carcinomatous picture

Differential diagnosis (1) Senile pruritus, (2) X ray dermatitis

Prognosis Children generally die within a year after diagnosis Only few cases can reach the adult age Infants die of marasmus whereas adults die of malignant changes in the skin

Treatment No treatment is of any use Sunlight should be avoided Verrucous lesions and Epitheliomas should be excised X ray and radium treatment are not indicated Vitamin A therapy in high doses (Arovit Roche) should be used for a long time

(b) Melanoma—Is a pigmented tumour of the skin composed of nævus cells (Fig No 162)

Varieties are —(1) Pigmented nævus, (2) Lentigo, (3) Melanocarcinoma, (4) Blue nævus

(1) Pigmented nævus are (i) Junction nævus and (ii) Intraepidermal nævus

Prognosis Is very grave in untreated cases Even in treated cases prognosis is not very good In the slow growing type occurring on face prognosis is not bad Persistent melanuria shows a grave prognosis

Treatment Prophylaxis in junction nævus is removal before puberty Any mole growing near the place of irritation should be removed particularly when a mole develops on the sole and in the subungual regions Benign moles are removed by carbon dioxide and by surgical excision

Curative is the deep and wide excision Regional lymphatics should be removed Since melanomas are radio resistant radiotherapy is useless

(c) Basal cell carcinoma—is also known as Rodent ulcer This is locally malignant and also a growing tumour of the skin

Etiology cause is not known May develop some times after arsenic ingestion

Age—No age is exempt but common after the age of 40 years Sex—Both sexes are equally affected

Signs and symptoms Types are —(i) Button type, (ii) Morphea type, (iii) Cystic type and (iv) Mixed type or Basi squamous type
Growth is very slow

Site—Centre of the face (Fig No 164) but the morphea like and the mixed type are commonly found on the trunk Nodular growth with central umbilication, rolled edge and pearly margin are the characteristic

patch of about half an inch in diameter on the buttock Histologically occurs in the dermis and do not undergo malignancy



Fig No 163
Melanocarcinoma Sole

Diagnosis (1) Clinical examination, (2) Histo pathology and special staining such as Fontana's stain will show melanin and Reticulin stain will show lattice net work in the tumour, (3) Histopathology—the loss of the *parallel arrangement* of the cells is seen The nucleus in the cell enlarges in size and mitosis *is present* Pigment *is found in abundance* both in the intercellular spaces and also intracellularly Elastic tissue is destroyed Activity in the *dermoepi* dermal nævus and lentigo whereas it is present slightly in junction nævus and is also present enormously *in* malignant melanoma

Differential diagnosis (1) Seborrhoic wart, (2) Sclerosing *hæmangioma*, (3) Lymphosarcoma, (4) Pigmented papilloma, (5) Blue nævus and (6) Granuloma pyogenicum



Fig No 166
Multiple pigmented basal cell carcinoma
(Case of Major B Chakraborty)

Diagnosis (1) An umbilicated nodular growth or a crusted growth in a person above 40 years of age, (2) Histopathology shows acanthosis in the mucosum Hyperplasia of the stratum basalis with mitosis of the nucleus in the cells There is no cell nest present (Fig No 167)

Differential diagnosis (1) Syphilis of the skin, (2) Lupus vulgaris (3) Squamous cell carcinoma and (4) Melanoma

Prognosis Good with treatment

Treatment (1) Excision, (2) Carbon dioxide freezing and (3) X ray therapy

(d) Squamous cell carcinoma—Is a rapidly growing cancer of the skin which forms cell nests and metastasize early

features Does not usually metastasize Some basal cell carcinomas are pigmented (Fig No 165 & 166)



Fig No 164
Basal cell carcinoma

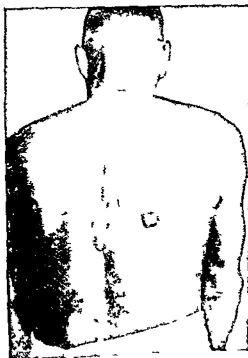


Fig No 165
Multiple basal cell carcinoma
(Case of Major B Chakraborty)



Fig No 166
Multiple pigmented basal cell carcinoma
(Case of Major B Chakraborty)

Diagnosis (1) An umbilicated nodular growth or a cru ted growth in a person above 40 years of age, (2) Histopathology shows acanthosis in the mucosum Hyperplasia of the stratum basalis with mitosis of the nucleus in the cells There is no cell nest present (Fig No 167)

Differential diagnosis (1) Syphilis of the skin, (2) Lupus vulgaris (3) Squamous cell carcinoma and (4) Melanoma

Prognosis Good with treatment

Treatment (1) Excision, (2) Carbon dioxide freezing and (3) X ray therapy

(d) Squamous cell carcinoma—Is a rapidly growing cancer of the skin which forms cell nests and metastasize early



Fig No 167
Histopathology of
Basal cell carcinoma

Etiology Is unknown Chronic irritation, X ray senile keratosis and cutaneous horn are predisposing factors *Age—middle age* *Sex—both sexes*

Signs and symptoms Single warty growth may occur on the face (Fig, No 168) Edge is everted (Fig No 169) and the growth fungates soon (Fig No 170 & 171) Regional lymph glands are affected early

Diagnosis (1) Single nodular growth, infiltrated at the base, (2) Site face, (3) Regional lymphatic glands are affected, (4) Histopathology concentric layers

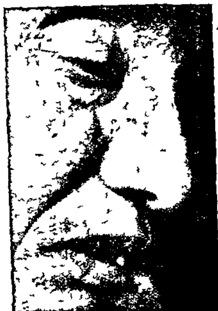


Fig No 168
Squamou cell carcinoma



Fig No 169
Epidermoid carcinoma
le ion on the nape of neck

of keratinized cells called cell nest are always found with hyperplasia of the ep dermis and mitosis

Differential diagnosis (1) Senile keratosis, (2) Lupus vulgaris and (3) Paget's disease

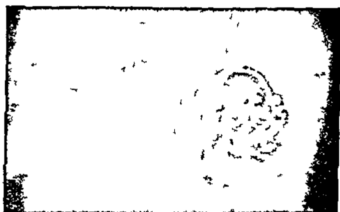


Fig No 170
Squamous cell carcinoma of Ear



Fig No 171
Squamous cell carcinoma

Prognosis In the early stage it is good but after metastasis has occurred the prognosis is grave

Treatment Excision in the early stage Both in the early and late stages application of radium is valuable X ray therapy is of great value

(e) Reticulosis—is the malignant change found in the reticulo endothelial system Reticulosis cutis includes (1) Leukæmia of the skin, (2) Hodgkin's disease of the skin, (3) Mycosis fungoidis of the skin and (4) Lymphosarcoma of the skin

Signs and symptoms In the early stage itching is most prominent followed by development of rashes which may be urticarial, eczematous and exfoliative This early picture may remain for weeks or months The colour of the skin in the tropics does not change very much except that it darkens In the late stage ulceration occurs

(1) Leukæmia cutis may be subdivided into (i) lymphatic, (ii) myelogenous, (iii) monocytic Leukaemia cutis may be seen at any age Skin lesions are tumours in the skin, subcutaneous haemorrhages even generalised herpes zoster and exfoliative dermatitis of very severe types are found associated

(2) Hodgkin s disease—occurs in both sexes Pruritus is the main feature Excoriated itchy papules develop all over the body Skin becomes ichthyotic, urticarial rash develops and even exfoliation occurs These symptoms continue for several months to years then the late stage supervenes when nodules appear which ulcerates accompanied with intermittent fever

(3) Lymphosarcoma Is divided into (i) large cell lymphosarcoma, (ii) small cell lymphosarcoma and (iii) Kaposi s multiple idiopathic pigmented sarcoma

Diagnosis (1) History, (2) Clinical examination, (3) Blood picture, (4) Histopathology shows infiltration in dermis and multiplicity of cells Dorothy Reed type of giant eell in Hodgkin's diease, pseudo-giant cell in mycosis fungoides Mitotic figures are also present There is multiplication of blood vessels in the dermis in lymphosarcoma

Differential diagnosis (1) Eczema, (2) Psoriasis, (3) Urticaria, (4) Exfoliative dermatitis due to arsenic, gold etc and (5) Pemphigus foliaceus

Prognosis *Is bad* *With* modern treatment *life may* be slightly prolonged

Treatment Arsenic by mouth as Fowler's solution in all stages Nitrogen mustard is helpful in certain cases Improvement in mycosis fungoides with Para aminobenzoic acid, in Kaposi's idiopathic sarcoma with ACTH have been observed Locally antipruritic lotions such as 1 p c Phenol in Lotio Calamine or 2% Menthol in ointment form are used X ray therapy is also helpful in some cases

CHAPTER XIII

COLLAGEN DISORDERS OF THE SKIN

There are some skin diseases in which the collagen fibres of the dermis are mainly affected

Classification (1) Lupus Erythematosus, (2) Scleroderma and (3) Dermatomyositis

LUPUS ERYTHEMATOSUS

Definition Is a chronic skin condition, sometimes becoming acute which is characterized by adherent scales on erythematous and atrophic base, with dilated mouth of the hair follicle pigmentation itching and is distributed primarily on the face, scalp, back, chest and extremities

Etiology Is fairly common in the tropics and form about 1 per cent of cases

Common causes are (1) Septic foci (2) Tubreculosis (3) Toxin of strepto and staphylococci, (4) Ovarian dysfunction as there is exaggeration of the skin lesion after each pregnancy and bafore menstruation in women (5) Allergic manifestation, (6) Due to exposure to the ultra violet rays of the sun

Varieties (1) Acute and (2) Chronic types Chronic type is subdivided into (3) Chronic discoid and (4) Chronic disseminate

Chronic discoid type of lupus erythematosus is quite common in the tropics and may form about 0.8 per cent of all skin cases Subacute and acute types are very rare in the tropics

Age—Commonly starts at the young adult age Sex generally seen in women but in the tropics both sexes are equally affected

Signs and symptoms The chronic discoid type of lesion may start as a circular pigmented spot anywhere on the face The spot increases peripherally and becomes pink in colour with a pigmented periphery There are greyish adherent scales with keratotic plugs on the under surface which fits in the dilated mouth of the hairfollicles The disease generally starts on both the malor regions (Fig No 172)



Fig No 172
Lupus erythematosus

and extends peripherally to get connected over the nose forming a butterfly-like lesion The wings of the butterfly lying on each malor region with the body on the bridge of the nose The lesion looks depressed from the surrounding skin due to atrophy

Site being the face, nose, back, chest and extremities Atrophy and depigmentation results after cure of the skin lesions Sometimes nodular growths may be found on the lower lip and chin The nodular growths are called *Lupus erythematosus hypertrophicus* Itching is the only symptom

During the year there may be exacerbation and quiescence of the condition Rarely ulceration occurs when and patient complains of burning sensation

The lesion may occur on the tongue as a white patch with a red halo The lip may be affected where the lesions are like white patches with red halo or there may be crusts with telangiectasia and swelling of lip (Fig No 173) May occur on scalp causing alopecia



Fig No 173
Lupus erythematosus
(lesion on both lips)

Lupus erythematosus lesion may be found on the conjunctiva where it is reddish and edematous in nature becoming atrophic and depressed later on Burning of the eyes may be the only symptom or there may be itching also

The chronic discoid lupus erythematosus becomes chronic disseminated The lesions on the face look inflammed and red macular lesions develope on the body There may be remission followed by relapse of the

skin condition within a month or two Sometimes the lesions may be urticarial, erythema multiforme or macular syphilide in types Rarely purpuric lesions may be seen Constitutional symptoms may be present

Systemic reactions - In the chronic discoid type there is no fever or malaise In the chronic disseminate type there may be malaise, fever, arthralgia In the acute disseminate type there is further exacerbation with loss of weight and lassitude There may be endocarditis associated with an acute disseminated lupus erythematosus when it is known as Libman Sacks disease

Diagnosis (1) In the chronic discoid type there may be one or many circular or oval or butterfly like lesions covered with adherent scales on erythematous base with a pigmented periphery associated with itching on the face, back and chest, (2) In the chronic disseminate or in acute disseminate types macular, urticarial or erythema multiforme type of lesions occur with inflammatory exacerbation of the facial lesions accompanied with malaise, irregular fever of unknown etiology and arthralgia and weakness

(3) Biopsy—Histopathology shows in the chronic discoid type thinning of the epidermis and presence of keratotic plugs at the mouths of the hair follicles Acanthosis of the st mucosum is seen with degeneration of the stratum basalis In the dermis there is destruction of the elastic tissue and collagen fibres with dense lymphocytic infiltration which runs parallel with the dermo-epidermal junction There is dilatation of the blood vessels and lymphatics In the acute disseminate type there is hyperkeratosis, atrophy of stratum mucosum and degeneration of the stratum basalis Dilatation of

the blood vessels and lymphatics with inflammation of the elastic and collagen fibers in the dermis with moderate lymphocytic infiltration, (4) Examination of Blood for total count of W B C, R B C, Differential count and Hæmoglobin per cent, (5) E S R—high, (6) blood W R is false positive, (7) Urine examination shows albuminuria, (8) L E cells and L E phenomenon are positive in acute lupus erythematosus only

Differential diagnosis (1) Seborrhoic dermatitis, (2) Lupus vulgaris, (3) Syphilis, (4) Alopecia areata and (5) Psoriasis

Prognosis Is favourable in chronic discoid type whereas in the chronic disseminate it is fair but the acute type ends in death in the tropics Alopecia is permanent

Treatment Prophylactic—for some people it is better to avoid the exposure to sun It is better to use a screening ointment while going out in the sun The ointment should consist of 1 per cent Para amino benzoic acid in vaseline Curative—(1) To investigate all the foci of infection and to treat them if found, (2) Bismuth—colloidal bismuth injections are given in doses of 0 2 gm biweekly for 10 weeks after urine examination Hygiene of mouth is important Orally bismuth may be given in dose of 75 mg of Bistrimate thrice daily (3) Crude liver extract may be injected intramuscularly in dose of 2 c c biweekly, 12 such, (4) Gold sodium thio sulphate is given by intravenous injections starting with 5 mg and increasing gradually by 1 mg every week until 30 mg is given, (5) Vitamin B_{12} in dose of 500 microgram can be injected intramuscularly every day to allay itching, (6) Atebrine orally has been used in dose of 25 mg

3 times daily for the first week, 25 mg 2 times daily for 10 weeks or more It is difficult *to carry the atebrine* treatment for its toxic effects such as yellow colouration of the skin, pain in the abdomen and atebrine psychosis

Locally may be used—(1) 1 p c Quinine bihydrochlor ointment, (2) 5 p c Para Amino Benzoic Acid ointment, (3) 2 p c Ung Hydrarg Ammon and (4) Lint Calamine

For subacute or acute disseminate types of cases (1) ACTH *aquous 40 units is injected intramuscularly* twice daily *for the first* week, ACTH *gel 40 units* is injected intramuscularly once daily for the second week Follow up treatment can be carried on by long acting ACTH, (2) Cortisone may be given by injection or by the oral route in dose of 25 mg tablet every 6 hours for the first week and then in graduated dose Sodium salt is not given but Potassium salt such as K salt (Calcutta Chemical) *is allowed during the corticotropine* and cortisone therapy, (4) Blood transfusion is helpful in the acute stage of the disease, (5) Vitamin C (500 mg), is injected intramuscularly twice daily, (6) *Multi* vitamin is given in high dosage by mouth, (7) Penicillin crystallin 'G' in aquous solution in dose of O 5 Mu daily is to be injected for any intercurrent infection, (8) Sulpha drugs are not used, (9) Quinine bisulphate may be given, (10) Oxygen inhalation is helpful

SCLERODERMA

Definition *Is a* skin disease *characterized by the* appearance of localized or generalized smooth, hard, white areas with a pigmented periphery on the skin which is red and swollen in the early stage later becoming atrophied and smooth

Etiology Varieties are found as (1) Localized type or morphœa and (1) Generalised type Cause is not known Acute infectious diseases, cancer, sunlight and psychological upsets may be responsible Women are commonly affected Found in the tropics Common amongst young adults

Signs and symptoms Localized or diffused swelling of the skin with redness Gradually these areas become smooth, whitish in colour, fixed to the underlying tissues and become atrophied Site—may be on the face (Fig 174 & 175) trunk, upper and lower extremities



Fig No 174
Scleroderma
(Morphea type on the forehead)

(Fig No 176) Commonly seen on hands which extends upwards and then on face Morphea often occurs on the extremities face and the middle line of the forehead Scleroderma of fingers is called



Fig No 175
Morphea
(Scleroderma on forehead)

sclerodactyle (Fig No 176 & 177) It is non-itchy and not tender Calcium deposition in different tissues is called *Calcinosis cutis* or Weissenbach syndrome



Fig No 176
Generalized scleroderma with sclrodactyle
(Case of Dr S C Desai)

Diagnosis (1) Localized or generalized inflammed skin or atrophied skin, (2) Sites—forehead or extremities or trunk, (3) Biopsy—histopathology shows flattening of the dermo epidermal junction with atrophy of the hair follicles and sweat-glands Degeneration



Fig No 177
Scleroderma with
sclerodactyle
(Case of Dr S C Desai)

and homogenization of the collagen fibers with the loss of parallel arrangement of the elastic fibers (Fig No 178), (4) Blood calcium level is high, (5) Creatine estimation is high (6) 17 Ketosteroid—low urinary Ketosteroid level

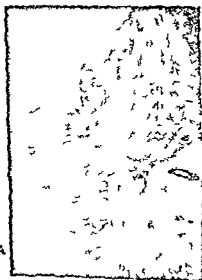


Fig No 178
Scleroderma
Histopathology

Differential diagnosis (1) Leucoderma, (2) Psoriasis

Prognosis Is fair in morphea as it may be cured even without treatment In generalized type patient dies of some intercurrent disease

Treatment Prophylaxis—nothing is known

Curative—(1) Physical therapy—such as massage, application of heat, (2) Para-Amino-Benzoic Acid in dose of 12 gram daily by mouth for about 12 weeks, (3) Thyroid extract by mouth gr½ daily, (4) Ditachysterol in dose of m 15 thrice daily by mouth, (5) Niacin and neostigmine 10 mg 2 to 3 times a day are helpful, (6) Penicillin injection is helpful, (7) Vitamin B complex in high dose is beneficial, (8) Cortisone can be given, (9) ACTH may also be injected during acute exacerbations Sympathectomy is sometimes helpful, (4) Glyceryl Trinitrate Ointment may be used locally

DERMATOMYOSITIS

Definition Is an acute or chronic skin disease characterized by inflammation of muscles and skin

Etiology Is unknown Sometimes coccal infection may be responsible Chronic systemic disease and visceral cancer may be the precipitating causes

Age—at any age Sex—both sexes

Signs and symptoms Weakness, pain and tenderness in muscles are common Muscles of the extremities are often first affected symmetrically (Fig No 179) In the early stage muscles are tender and normal in

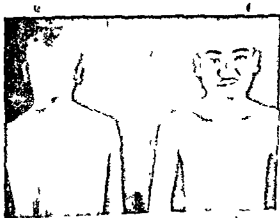


Fig No 179
Dermatomysitis
(Case of Dr S C Desai)

shape but later on the muscles become hardened Contracture may result Respiration and swallowing may be very difficult Enlargement of spleen and lymphatic glands may be associated There are reticulated erythematous plaques on the trunk, extremities and on the dorsum of the hands

Diagnosis (1) Progressive weakness in muscles resulting in hardening of muscles and difficulty in respiration and swallowing (2) E S R—high, (3) Blood—serum estimation shows high globulin, (4) Urine analysis shows creatinuria (5) Biopsy—histopathology shows inflammation of muscle fibers and infiltration with polymorphonuclear cells Proliferation of connective tissues and hyalinization of collagen fibers in the dermis

Differential diagnosis (1) Lupus erythematosus and (2) Scleroderma

Prognosis Death may result in few weeks in acute but when recovery occurs there is muscular weakness left In chronic cases patients suffer long and the disease may become stationary

Treatment Prophylaxis—is not known Curative (1) Removal of infective foci (2) Auto vaccine therapy may be helpful and non specific protein therapy with milk injection may be tried, (3) Penicillin therapy is sometimes indicated, (4) Vitamin E gives some beneficial effect (4) Cortisone—has been found to be helpful in some cases (5) ACTH is also helpful, (6) Androgen therapy has been rarely helpful, (7) Antihistamin drug may be used, (8) Blood transfussion is advocated (9) Rest in bed is essential (10) Diet—high protein diet should be given consisting of meat fish, egg and for the vegetarians milk and channa

CHAPTER XIV

SEBORRHOEIC DISORDERS OF THE SKIN

Seborrhoeic disorders include (1) Acne vulgaris, (2) Acne rosacea and (3) Seborrhoea

ACNE VULGARIS

Definition Acne vulgaris is a chronic inflammation of the pilo sebaceous follicle accompained with comedone formation and seborrhoea particularly of the face during adolescence

Etiology (1) Endocrine seen commonly in adolescents and increases during menstruation Is supposed to be due to the imbalance of oestrogen and androgen Predominence of androgen has been observed in acne vulgaris Sex glands—when there is an excessive production of androgen upsetting the endocrine balance with estrogen it results in the production of acne vulgaris There is an exacerbation in women during menstruation, (2) Metabolic disturbance—as in thyroid deficiency, (3) Hypovitaminosis A and C—low vitamin intake causes acne vulgaris, (4) Diet—in some patients particular *diet causes* aggravation of acne *such as* carbohydrate rich *and fatty food*, (5) *Sex—found* in both sexes, (6) Age—common at the adolescent age but may be found in children and also in women during pregnancy *Common age is 13 to 30 years*

Signs and symptoms Lesions are mainly papules, nodules and pustules situated on the face

(Fig No 180) shoulders and chest There may be inflammation present with the papular type but



Fig No 180
Acne vulgaris
(showing black heads all over the back)

nodular types are always accompanied with inflammation The pustular lesions of acne vulgaris are said to be due to the entrace of pyogenic organisms in the hair follicles The important lesion in acne vulgaris is the formation of comedo caused by the formation of excessive keratinization at the mouth of the hair follicle The acne may be cyst like when it is called *cystic acne* (Fig No 181) Scalp shows excessive amount of oily secretion called seborrhoea Sometimes the face is also oily A type of acne has been described as the *tropical acne* Tropical acne is seen only in foreigners visiting tropics and is characterised by the exacerbation of the ordinary acne vulgaris



Fig No 181
Acne cystica
(Case of
Dr B S Verma)

Diagnosis (1) Age of patient —adolescent age (2) Papular or pustular eruption with black heads on face, back chest and shoulders

CHAPTER XIV

SEBORRHOEIC DISORDERS OF THE SKIN

Seborrhoeic disorders include (1) Acne vulgaris, (2) Acne rosacea and (3) Seborrhoea

ACNE VULGARIS

Definition Acne vulgaris is a chronic inflammation of the pilo sebaceous follicle accompanied with comedone formation and seborrhoea particularly of the face during adolescence

Etiology (1) Endocrine seen commonly in adolescents and increases during menstruation Is supposed to be due to the imbalance of oestrogen and androgen Predominence of androgen has been observed in acne vulgaris Sex glands—when there is an excessive production of androgen upsetting the endocrine balance with estrogen it results in the production of acne vulgaris There is an exacerbation in women during menstruation, (2) Metabolic disturbance—as in thyroid deficiency, (3) Hypovitaminosis A and C—low vitamin intake causes acne vulgaris, (4) Diet—in some patients particular diet causes aggravation of acne such as carbohydrate rich and fatty food, (5) Sex—found in both sexes, (6) Age—common at the adolescent age but may be found in children and also in women during pregnancy Common age is 13 to 30 years

Signs and symptoms Lesions are mainly papules, nodules and pustules situated on the face

(Fig No 180) shoulders and chest There may be inflammation present with the papular type but



Fig No 180
Acne vulgaris
(showing black heads
al over the back)

nodular types are always accompanied with inflammation The pustular lesions of acne vulgaris are said to be due to the entrace of pyogenic organisms in the hair follicles The important lesion in acne vulgaris is the formation of comedo caused by the formation of excessive keratinization at the mouth of the hair follicle The acne may be cyst like when it is called *cystic acne* (Fig No 181) Scalp shows excessive amount of oily secretion called seborrhoea Sometimes the face is also oily A type of acne has been described as the *tropical acne* Tropical acne is seen only in foreigners visiting tropics and is characterised by the exacerbation of the ordinary acne vulgaris



Fig No 181
Acne cystica
(Case of
Dr B S Verma)

Diagnosis (1) Age of patient —adolescent age (2) Papular or pustular eruption with black heads on face back, chest and shoulders

with inflammation and oiliness of the face and scalp, (3) Histopathology—perifolliculitis with follicular plug of sebum

Differential diagnosis (1) Acne rosacea, (2) Oil acne, (3) Tar acne, (4) Acne due to bromide and iodide, and (5) Pustular syphiloderm

Prognosis Acne vulgaris troubles a person between the age of 13 to 30 years Prognosis is good in acne vulgaris but in acne cystica sinuses, scars and keldoids usually develop

Treatment (1) Diet is most important Normal diet is given including meat, fish, milk, vegetable, rice and bread Fatty food is cut down to the minimum Carbohydrate intake should be restricted, (2) Exercise—walking, swimming, joining outdoor games are essential Sedentary habit is bad in acne patients, (3) Locally—Bland soap should be used regularly and particularly face should be washed before going to bed The following lotion may be applied every night in mild cases of acne vulgaris (1) Zinc Sulph—gr 25, Potassium Sulphuratum—gr 15, Aqua Rosae—dr 1, Aqua distilata—oz 1 Ft lotio To apply every night (2) An ointment may be rubbed every night after washing the face with a bland soap and warm water The rubbing of the following ointment should be done *for 3 nights followed by rest for one night* and repeating 6 times Resorcin—gr 5, Acid Salicylic—gr, 10, Vaseline alba—oz 1 or Sulpher ppt—gr 10, Acid Salicylic—gr 10, Vaselin alba—oz 1

(c) X ray therapy or ultra-violet therapy may sometimes be helpful, (4) Staphylococcal toxoid vaccine

may be given in pustular acne in gradually increasing doses, (5) Antibiotic—penicillin crystalline 'G' is often helpful in pustular acne vulgaris, (6) Shampooing of the scalp is helpful with Selsun (Abbott) when there is seborrhoea present, (7) Endocrine therapy—some patients do well with oestrogen therapy In females it is given for 14 days starting 14 days before the expected menstruation and the drug should be stopped with the appearance of menstruation Oestrogen may also be carefully given even to the males and the patient must be watched for the development of gynecomastia Thyroid in small doses (gr ½ daily) is sometimes helpful in acne vulgaris (8) Vitamin therapy—Vitamin A in high doses (Arovit Roche 100 000 I U) daily for a period of 6 months with vitamin C are helpful, (9) Anaemia when associated in the tropics should be treated with iron therapy and constipation must be corrected, (10) Acne scars may be treated by Carbon dioxide snow application

ACNE ROSACEA

Definition Is a chronic inflammation of the pilo sebaceous follicles of the center of the face

Etiology (1) Gastrointestinal troubles are commonly associated, (2) Psychological maladjustment may be responsible (3) Vitamin B_2 deficiency may be found

Signs and symptoms Redness of the center of the face with hot curries, strong and hot tea, coffee, alcohol and after gastro intestinal upset and during menstruation are the early signs Later on the redness becomes permanent with papules and pustules on the chin nose, malor regions and on the forehead

with inflammation and oiliness of the face and scalp, (3) Histopathology—perifolliculitis with follicular plug of sebum

Differential diagnosis (1) Acne rosacea, (2) Oil acne, (3) Tar acne, (4) Acne due to bromide and iodide, and (5) Pustular syphiloderm

Prognosis Acne vulgaris troubles a person between the age of 13 to 30 years Prognosis is good in acne vulgaris but in acne cystica sinuses, scars and keldoids usually develop

Treatment (1) Diet is most important Normal diet is given including meat, fish, milk, vegetable, rice and bread Fatty food is cut down to the minimum Carbohydrate intake should be restricted, (2) Exercise—walking, swimming, joining outdoor games are essential Sedentary habit is bad in acne patients, (3) Locally—Bland soap should be used regularly and particularly face should be washed before going to bed The following lotion may be applied every night in mild cases of acne vulgaris (1) Zinc Sulph—gr 25, Potassium Sulphuratum—gr 15, Aqua Rosae—dr 1, Aqua distilata—oz 1 Ft lotio To apply every night (2) An ointment may be rubbed every night after washing the face with a bland soap and warm water The rubbing of the following ointment should be done for 3 nights followed by rest for one night and repeating 6 times Resorcin—gr 5, Acid Salicylic—gr, 10, Vaseline alba—oz 1 or Sulpher ppt—gr 10, Acid Salicylic—gr 10, Vaselin alba—oz 1

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ACNE ROSACEA

Definition Is a chronic inflammation of the pilo sebaceous follicles of the center of the face

Etiology (1) Gastrointestinal troubles are commonly associated, (2) Psychological maladjustment may be responsible, (3) Vitamin B_2 deficiency may be found

Signs and symptoms Redness of the center of the face with hot curries, strong and hot tea coffee, alcohol and after gastro intestinal upset and during menstruation are the early signs Later on the *redness* becomes permanent with papules and pustules on the chin, nose, malor regions and on the forehead

Gradually the tip of the nose becomes enlarged and is called *Rhinophyma*

Diagnosis (1) History of chronic gastro intestinal upset or psychological troubles, (2) Flushing of the face after hot food or during menstruation, (3) Papules and pustules on the centre of the face, (4) Riboflavin deficiency in the eyes, mouth etc (5) Histopathology shows dilatation of the dermic vessels and infiltration of the pilosebaceous follicles with small round cells, (b) Gastric analysis shows achlorhydria or hyperchlorhydria

Differential diagnosis (1) Acne vulgaris, (2) Drug rash, (3) Secondary syphilis, (4) Tuberculosis cutis

Prognosis Fair with treatment

Treatment Diet should be regulated and gastro intestinal troubles should be treated with Riboflavin injection and Vitamin B-complex orally *Acid Hydro* chlor dil in doses of 1 1 10 soon after food is helpful If a psychological trouble is causing the disease some sedative is given such as phenobarb gr $\frac{1}{2}$ at night for 3 to 4 nights or bromide in mixture with valerian for several nights Psychotherapy is helpful

Locally lotio Calamine is useful Sometimes 1 p c Resorcin ointment rubbing helps As a routine Ung Sulpho salicylic is rubbed twice daily Superficial X-ray therapy is also indicated Rhinophyma is treated by plastic surgery

SEBORRHOEA

Definition Is a constitutional recurrent skin disease characterised by inflammation and scaling which

starts from the scalp and spreads down the face, neck, trunk and the limbs

Etiology It is much less prevalent in the tropics Sebaceous saretion is influenced by emotional factors, digestive disturbances infections nutritional deficiencies and by external causes such as chemical The disturbance in the sebaceous secretion produces the seborrhoeic condition

Classification (1) Seborrhoic capitis is the sebor hoea of the scalp and (2) Seborrhoeic dermatitis is the seborrhoea of the trunk May occur at any age but commonly at puberty and affects both sexes

Signs and symptoms In children blackish crusts are found on scalp and behind the ears and groins Scaly eczematous lesions may involve the face and scalp (Fig No 182) in an adult which is rarely seen in an infant Flexure surfaces are commonly involved

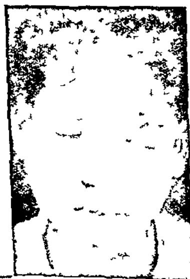


Fig No 182
Seborrhoea

such as as the axilla and crural regions The scalp, the forehead, eyelids, ala nasi, meatus of the ear, face, even lips may be involved as also the axillæ, intra scapular regions, front of the chest down to the pubic region Even palms and soles may develop pompholyx type of lesion in seborrhoea Genitalia may be affected in both sexes with seborrhoea There may be oozing, crusting or only dry scaling Sometimes erythematous patches may be seen The seborrhoic lesions are itchy Blepharitis, melanoderma, alopecia, seborrhoic wart in old age may develop as complications

Diagnosis (1) Itching, oozing or scaling dermatitis, (2) Typical distribution, (3) Familial history, (4) Histopathology shows hyperkeratosis and parakeratosis of the stratum corneum There is acanthosis and intra cellular edema in the stratum mucosum Dermis shows dilatation of the papillary vessel *with* perivascular lymphocytic infiltration, (5) Blood sugar may be high normal or may be often hyperglycaemic

Differential diagnosis (1) Psoriasis of the scalp is differenciated by matting of the hair with sebum *in* seborrhoic capitis but histopathologically there is no difference, (2) Eczema of scalp is difficult to diagnose histopathologically but in seborrhoea there is typical seborrhoic distribution if it spreads down the trunk, (3) Ringworm of the scalp is diagnosed by microscopical examination of the infected hair, (4) Infantile Eczema—generally starts on the cheeks as grouped vesicles which soon starts oozing whereas the seborrhoea starts on the scalp which extends down the face, neck and trunk, (5) Pityriasis versicolor by the microscopical examination where microsporon *furfur*

is found, (6) Dermal Leishmaniasis in the depigmented stage by the history of having had Kala Azar and by the histopathological examination of the skin for Leishman—Donovon bodies, (7) Pityriasis rosea is differential by its seasonal occurrence and typical bathing trunk distribution (Genji and underwear) and pink oval lesions along the clevage line of the ribs with scales at the periphery looking towards the centre

Prognosis Is good with treatment but is never cured

Treatment General patient should avoid sedentary habit and should take regular exercise Bowels must be kept moving Worry and anxiety must be avoided as far as possible Diet should be mixed type with vegetables Milk should be avoided Alkali is given to keep urine alkaline in the acute stage Vitamins are of particular value like Vitamin B complex Vitamin A and Vitamin C Sedative is required in the acute stage such as pheno barb gr ½ twice daily Methionine (Neomethidine—Neo Phama) is helpful

Locally a shampoo should be used for the scalp like Selsun (Abbott) or a shampoo containing Liq Picis Carb det —dr 1, Oil Recini—dr 1 Liquid soap—oz 2 After washing scalp with shampoo a lotion may be used for the scalp containing Resorcin—gr 2 Liq Picis carb det—m 4 50 p c Spirit Rect—ad oz 1 For the scalp an oil free shampoo is used 2 to 3 times a week followed by the application either of lotion or ointment Acid Salicylic—gr 10 Sulpher ppt—gr 10, Oil of cade—m 10 Vaseline alba—oz 1 ung for external use Locally in the acute stage for the body Argenti Nitras—gr 5, Aqua destil—oz 1 Ft lotio supply oz 8 in a coloured phial To apply every hour

for one day followed by on the 2nd or 3rd day a lotion containg Sulpher ppt —gr 10, Calamine ppt—dr 1 Aqua Distil—oz 10

In the chronic stage an ointment is used for the body Sulpher ppt —gr 10, Acid Salicylic—gr 10, Vaseline alba - oz 1 Ft Unguentum Use ointment twice daily for the body or scalp after bath with bland soap

PSORIASIS

Definition is a chronic noninfectious skin disease characterised by silvery scales on erythematous base with itching and distributed mainly round the elbow, round the knee, sacral region and on the scalp

Etiology Is not known yet It is quite a common disease in the tropics It forms about 3 per cent of all skin cases in India It affects both sexes Cases of psoriasis have been found in patients between the age of 3 months to 90 years but is commonly found in adults Metabolic disorders are sometimes held responsible for its development Endocrine disorders, vitamin C deficiency with higher vitaminosis A, stress and strain and psychosomatic factors are said to be responsible Septic foci are responsible predisposing factors and has often preceeded an eruption of psoriatic lesion Sometimes psoriasis and arthritis are associated Climate may have some influence and psoriasis cases are commonly seen in winter months in the tropics

Signs and symptoms Lesion of psoriasis may start as a macule which is reddish in colour Silvery white scales appear on the macule Macules may become papules after a time When a macule or a papule is scraped

by a pointed instrument scales on either side of the line appear and this test is called *tache de bouge* On scraping bleeding points appear Distribution is on the extensor sufaces Lesions may be found on the scalp on the trunk, elbows (Fig No 183) sacral and gluteal



Fig No 183
Psoriaeis forearm
(Case of Major N R Gupta)



Fig No 184
Psoriasis
(Acuts aczemators type)

regions knees (Fig No 184) umbilicus and on the nails of fingers and toes Psoriatic lesions may be found on palm and sole also and is known as palmer or planter psoriasis or pustular psoriasis Lesions may develop along the line of a scratch on the skin which is called *Koebner's phenomenon*

Varieties of psoriasis (1) Psoriasis punctata lesions look like small points, (2) Psoriasis guttata lesions look like rain drops (Fig No 185), (3) Psoriasis nummularis lesions look like coins, (Fig No 186)

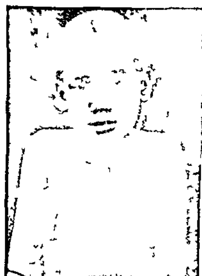


Fig No 185
Psoriasis
Guttate type
(Case of Dr R P Gupta)



Fig No 186
Psoriasis
(Case of Dr K C Sahu)

(4) Psoriasis circinata lesions are in patches and form polycyclic figures, (5) Posiasis figarata lesions are like figures, (6) Psoriasis follicularis lesions affecting hair follicles, (7) Psoriasis arthropathica lesions are associated with arthritis (8) Pustular psor asis erythemato pustular lesions appear on the instep of sole and on the palm, (9) Eczematous psoriasis, (10) Psoriasis seborrhoea and (11) Flexural psoriasis

Diagnosis (1) Typical lesion silvery scales on erythematous base distributed on scalp, extensor sur faces elbows, knees and sacral regions and also on the nail (2) Tache de Bouge test positive, (3) Koebner's phenomenon positive, (4) Biopsy and histopathology Hyperkeratosis, parakeratosis elongation of the rete pegs, absence of stratum granulosum thinning of rete mucosum above the papillae, oedema of the papillary body and the presence of microabscesses of Munro

Differential diagnosis (1) Eczema (3) Seborr hoic dermatitis (3) Squamous syphilis (4) Pityriasis rosea (5) Lichen planus, (6) Lichen simplex chronicus (Widal), (7) Tinea corporis, (8) Acrodermatitis perstans Acrodermatitis tropicalis and pompholyx should be differentiated from pustular psoriasis

Prognosis Condition can be improved with treat ment and patient may be made temporarily free but permanent cure is not pos ible Relapses are common

Treatment General (1) Any systemic disease should be treated and septic foci should be removed, (2) Low protein diet is advisable Vegeterauian diet with milk is commonly recommended Vitamin C rich and low vitamin A diet is helpful, (3) Internally arsenic

by mouth such as Fowler's solution in dose of one drop thrice daily for a long time and the dose is often increased gradually, (4) Vitamin C in high dose (500 mg I M twice daily) during the acute stage and in small dose (500 mg orally daily) are advocated during the chronic stage Other vitamins such as Vitamin A and Vitamin D are sometimes helpful ACTH in high dose *during the acute stage and in small dose for the follow* up is a helpful adjuvant to the therapy but ACTH is never a specific for psoriasis There is frequently an exacerbation in psoriasis after this therapy but is only helpful in prosiasis arthropathica Cortisone should never be given in psoriasis because when it is stopped the relapse is very severe

Locally during the acute stage 1 p c Ung Ichthyol (Ichthyol Gr 5 in Vaseline alba oz 1) During the subacute stage Ung Hydrarg ammon (Hydrarg ammoniata gr 20 in Vasiline oz 1) alone or with 2 p c Acid Salicylic are used

During the chronic stage 2 p c Ung Chrysarobin gradually increasing to 5 p c for lesions localised on body except face and scalp is used otherwise irritation may give rise to acute conjunctivitis For the scalp 2 to 5 p c oil of cade in vaseline together with 2 p c acid salicylic

For ambulatory patients painting with crude coal tar is very helpful but should not be applied long as it is one of the carcinogenics

Electrotherapy generalised ultra violet exposure

Superficial X ray therapy—In generalised eruption 40r with 75 KV is valuable but for small isolated patches 80r with 75 KV is recommended For pustular

psoriasis and for the psoriasis of nails 6 to 8 weekly exposures of superficial X ray therapy in dose of 80 r with 75 KV are given Thorium X solution (1,500 Electrostatic unit per ml of solution) may bs locally applied on chronic lesions anywhere on the body

LICHEN PLANUS

Definition Is a chronic skin disease characterized by reddish brown papules or blackish red hyperkeratotic plaques on the skin of the front of the wrist and other parts of the body including the mucous membranes of mouth tongue glans penis and vagina

Etiology It is not an infectious disease Both sexes are affected equally Age—common in adults but no age is exempt No cause is known but worry seems to play some part in its development It forms about 2 p c of all the skin cases in the tropics

Signs and symptoms May start with itching pin point shiny lesions on the skin or as one or two very small polygonal papule reflecting light infront of both wrists or both shins Distribution is symmetrical over the flexor surfaces forearms wrists (Fig No 187), shins ankles (Fig No 188) but sometimes seen only on one side of the body The polygonal papules may coalesce and form a big sheet on the trunk when it is called *Lichen pl nus disseminatus* (Fig No 189) when papules become very prominent and form a plaque commonly on the front of the shins it is known as (Fig No 188) *Lichen planus hypertrophicus* Rarely seen like a band on one side of the body and is called *Lichen linearis* Sometimes whitish oval spots are seen called

Lichen planus atrophicus These spots are conglomeration of polygonal atrophic macular shiny areas When seen with a lens a dimple may be seen on each of the papules and it represents the opening of the hair follicle Fine white points and white lines



Fig No 187

Lichen Planus

may be seen on the lesions with a magnifying glass and are known as *Wickham's striæ* Lichen planus

of skin heals after leaving pigmentation but occasionally white atrophic looking spots are left These black or



Fig No 188
Lichen planus
hypertrophicus
(Case of Dr N C Sanyal)



Fig No 189
Lichen planus
disseminatus

white spots disappear ultimately Rarely lichen planus affects one of the limbs as a band such as the inferior extremity from heel to the groin This is a slowly forming skin disease taking several months to develop with itching and typical Koebner's phenomenon

Diagnosis (1) Polygonal flat topped itching papules which are shiny and situated on the front of th wrist, forearm, ankle and leg and are symmetrical

Lichen planus atrophicus These spots are conglomeration of polygonal atrophic macular shiny areas When seen with a lens a dimple may be seen on each of the papules and it represents the opening of the hair follicle Fine white points and white lines



Fig No 187

Lichen Planus

may be seen on the lesions with a magnifying glass and are known as *Wickham's striæ* Lichen planus

of skin heals after leaving pigmentation but occasionally white atrophic looking spots are left These black or



Fig No 188
Lichen planus
hypertrophicus
(Case of Dr N C Sanyal)



Fig No 189
Lichen planus
disseminatus

white spots disappear ultimately Rarely lichen planus affects one of the limbs as a band such as the inferior extremity from heel to the groin This is a slowly forming skin disease taking several months to develop with itching and typical Koebner's phenomenon

Diagnosis (1) Polygonal flat topped itching papules which are shiny and situated on the front of the wrist, forearm, ankle and leg and are symmetrical

in distribution and may be present on the mucous membrane of the mouth as white patchy lesions, (2) Lesions may develop on a scratch—positive Kœbner's phenomenon, (3) Histopathology—Hyperkeratosis, thickening of the stratum granulosum and acanthosis Degeneration of the dermo epidermal junction Round cell infiltration in the dermis

Differential diagnosis (1) Psoriasis, (2) Pityriasis rosea, (3) Discoid lupus erythematosus, (4) Papular syphiloderma, (5) Striate lichen planus from lichen striatus

		Striate Lichen planus	Lichen Striatus
1	Koebner's phenomenon	present	absent
2	Itching	present	absent
3	Histopathology	like lichen planus	like eczema

Prognosis Is a chronic skin disease Generally it is completly cured but may relapse

Treatment General treatment—Improvement of general health Psychosomatic troubles should be taken into account and change of surroundings is advisable Tonic containing vitamin B complex is valuable

Internally Arsenic (Stovarsol—25 mg tablet), vitamin B-Complex and Enesol (Comar cie) injected I M, Bismuth (0 2 gm) is given by intramuscular injection and can be repeated after a month's interval Mercury as Hydrag perchlor gr 1/16 per dose, three times daily orally and locally 4 p c Ung Hydrag Ammon are used as a routine. Treatment of lichen planus is mercury in and mercury out X ray therapy locally is valuable In a localised lesion 80 r weekly with 75 KV is given for 4 to 6 times at weekly intervals

but in gensralised or in acute cases 40 r at 95 KV in one or two doses are recommended

PITYRIASIS ROSEA

Definition Is an acute skin disease characterised by erythematosquamous patchy lesions distributed on the trunk arms and thighs with slight itching

Etiology It is a mildly infectious acute skin disease Cause is not known Virus, internal toxin and treponema may be responsible Quite common in the tropics and forms about 0 1 p c of skin cases It is seasional Seen at the chage of seasions Affects all ages and both sexes

Signs and symptoms An Erythemato squamous circular lesion about an inches in diameter which appears on the pectoral scapular, umbilical or inguinal regions This patch is not itchy and is called the *herald patch* Herald patch is present in one third of all cases of pityriasis rosea 7 to 8 days after the appearance of the herald patch small oval reddish rash appear on the body while the herald patch gets almost cleared up by that time The lesions are papular red spots becoming oval reddish patches of one fourth inch in size Scales are arranged at the periphery which look towards the centre As the lesions grow old the reddish colour changes into brownish red to brown and ultimately when healed look like depigmented spots Seen over the trunk and symmetrically down the arms upto the elbow and down the thighs as far as the knees but may be generalized also Face is not affected The rash persists for only six weeks and is slightly itchy Sometimes sore throat preceeds the rash

Diagnosis (1) Seasional appearance, (2) Typical symmetrical distribution and on the areas covered by the underwears (Genji and underpant), (3) Rose red colour of lesions with scales arranged at the periphery and lesions arranged along the cleavage line of the ribs, (4) Biopsy and histopathological examination shows—parakeratosis and acanthosis, oedema in the dermis and migration of cells in the epidermis

Differential diagnosis (1) Ringworm, (2) Pityriasis versicolor, (3) Psoriasis, (4) Seborrhoic dermatitis, (5) Macular syphilide, (5) Leprosy, (6) Dermal leishmaniasis

Prognosis Good even without any treatment

Treatment No treatment is usually necessary Lotio Condy's (Pot Permanganate gr 20 in Aqua oz 1) bath daily followed by 2 p c Ung Acid Salicylic Sometimes generalised ultra violet therapy clears all the lesions by only one exposure Antibiotic like aureomycin, terramycin, chloromycetin, Ilotycin may be used for 4 days orally with vitamin B complex

AINHAM

It also known as Dactylolysis spontanea

Definition Is a chronic skin disease occurring specially in the tropics and is characterized by the formation of a fibrous ring around the little toe of one or both feet The ring gradually narrows and in several weeks to several years the toe falls off

Etiology Is unknown Specially seen in the tropics

Types—(1) Ainham and (2) Pseudo ainham

Signs and symptoms A groove generally appears *on the side or dorsum of one of the* little toes (Fig No 190) which as it deepens it also encircles the toe and

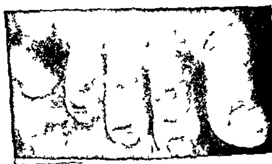


Fig No 190
Ainham

the time taken may be several weeks The depression is formed by a fibrous ring which gradually narrows and cuts the toe without pain or bleeding The underlying bone also gets thinned but there is no osteomyelitic change or rarefaction seen Sometimes it effects the little toe of the other foot (Fig No 191) in

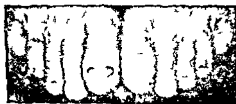


Fig No 191
Ainham

several weeks to several years after the affection of the former toe Rarely the third and even the fourth

toe of the other foot also is affected There is no discharge or signs of inflammation below the condition Histoperthology of the fibrous ring shows thickening of all the layers of the epidermis (Fig No 192) There



Fig No 192
Histopathology of Ainham
(Biopsy of the fibrous constriction)

is no systemic reaction The tarsal bone gives way and the toe falls off Radiologically there is thinning of the tarsal bone (Fig No 193) Pseudoainham is either congenitally absent toe finger or limb or is due to other diseases like leprosy, scleroderma

Differential diagnosis (1) Leprosy, (2) Chronic ulcer

Treatment Prophylaxis—to use foot wear

Curative—cutting the bands at different places

Cauterising with Iodine Penicillin injection for 2 weeks Warm condy s bath

POROKERATOSIS (MIBELLI)

Definition Is a chronic disease characterised by keratotic ring shaped lesions occuring on the body

Etiology No cause is known It is a hereditary keratodyskeratosis It is supposed to be due to vitamin A deficiency Is found in the tropics Men are commonly affected Age starts generally at the prepubertal age and stays throughout life Forms 0 1 p c of skin cases



Fig No 193
Sklagram of left foot with Ainham of toe
(Narrowing of bone of the little toe)

Signs and symptoms Sometimes it is itchy Usually there is no symptom at all The lesions may be very few in number (Fig No 194) or starts insidiously over a sweat duct opening hence called "poro as a keratotic follicle or [keratotic papule This papule enlarges in size and gets

depressed in the centre until a keratotic ring is formed and is described as kerato atrophoderma In size the largest



Fig No 194
Porokeratosis (Mibelli)
(Lesions on latrel aspect of the right Knee joint)

one may be half an inch in diameter with a black keratotic ring enclosing an atrophied skin The extension is centrifugal The hair, sweat and sebaceous glands on the atrophied skin undergo atrophy Lesions are multiple but rarely single May occur all over the body on the skin and rarely on the mucous membrane

Diagnosis (1) The typical keratotic ring like balckish lesions, (2) Sites on the dorsum of hands, buttocks, thighs are common places, (3) Biopsy—histopathology shows hyperkeratosis of all the layers of the epidermis Corps ronds are found in the epidermis In the dermis there is atrophy of the glands (Fig No 195)

Differential diagnosis (1) Basal cell carcinoma and (2) Senile keratosis

Prognosis Is not curable

Treatment Massive vitamin A therapy (Arovit Roche tablet 100,000 i u) is given by mouth for a long time Excision of the lesions may be done



Fig No 195
Porokratosis (Mibelli)
Histopathology

MILIARIA RUBRA

This is commonly known as prickly heat

Definition Is a tropical skin disease characterized by the formation of erythematopapular rash with pruritus

Etiology High humidity with tropical heat is supposed to be responsible for the causation of prickly heat Low vitamin A nutrition is responsible Due to the maceration of the stratum *corneum* the sweat ducts get blocked and thus the rash is produced from each of the blocked sweat duct Common amongst young adults but may be found in any age and in both sexes

Signs and symptoms Erythemato papular rash found during summer and monsoon later becoming papulo vesicular and some becoming pustular Site-back, chest and flexure surfaces and sometimes the face is also affected but palm and sole are never affected Covered areas liable to friction are generally affected It is very pruritic When the sweat is retained and causes the dilatation of the ducts half pea sized multiple lesions particularly appear on the face which are known as *summer boils*

Diagnosis (1) Erythemato papulo vesicular lesions on the covered areas of the body during the summer and monsoon months in a tropical country, (2) Itchy rash becoming squamous when the condition subsides leaving no scar, (3) Pilosebaceous follicles are free whereas the sweat ducts are involved, (4) Histopathology—blockage of the sweat duct with a keratotic plug Intraepidermal part of the sweat duct is destroyed Dermis shows edema with small round cell infiltration and dilatation of papillary vessels

Differential diagnosis (2) *Sudamina*, (2) *Seborrhoea*

Prognosis Good

Treatment Prophylaxis is to avoid hard labour during the day in summer and to use electric fans Daily bath with cold water twice daily Use of loose garments is a good prophylaxis in the tropics Hot tea and pickles in the food should be avoided Vitamin A should be taken in dose of 50,000 i u twice daily

Curative—Application of lotio *calamine is helpful* In the generalized type Vitamin A internally is helpful

CHAPTER XVI

METABOLIC DERMATOSIS

There are some skin diseases which are due to the disturbance in the mineral metabolism such as Calcium, Magnesium, Potassium Sodium in the body Calcium is most important as it is used in high dose in the form of Calciferol in the treatment of tuberculous infection of the skin Disturbance in protein metabolism has been observed in several diseases such as pemphigus and dermatomyositis Disturbance in lipid metabolism in psoriasis, carbohydrate metabolism in diabetes mellitus and prophyrin in porphyria

CALCINOSIS CUTIS

Calcium deposition in the body may be (1) Localised, (2) Generalised and (3) Metastatic

Local deposition of calcium in the skin is found secondary to various diseases such as scleroderma, milia, sebaceous cysts Generalised calcinosis cutis is supposed to result from circulatory insufficiency The metastatic calcinosis cutis is due to the hyperparathyroidism and is due to excessive intake of Vitamin D Ulceration may develop to excrete the calcarius material Normal blood calcium level is from 9 to 11 mg per 100 ml blood Normal blood calcium level does not *signify* normal calcium metabolism

Prognosis Is good in the localised and metastatic types whereas the generalised type is fatal

Treatment There is no treatment for the generalised calcinosis cutis In the localised type excision of the plaque with cutting down the intake of calcium and to stop the calcium therapy Excising the plaque is also advocated

PORPHYRIA

It is also known as porphyria cutanea tarda

Definition Is a chronic skin disease due to the defect in the porphyrin metabolism

Etiology It is found in the tropics but is not a common ailment It is due to an error of porphyrin metabolism Affects all ages and both sexes Types are (1) Congenital, (2) Acquired and (3) Secondary

Signs and symptoms In the congenital type the skin shows photosensitivity to light Bullous rash is found on the face and *exposed parts of the body Lesions* become eczematous and heals leaving pigmentation

When affects the skin of infants and children it is the congenital type but when the skin of the adults only are affected it is another variety of congenital type known as *Prophyria Cutanea Tarda* In this adult type the patient is commonly a female with hirsuitism The patient may have intermittent abdominal pain

Diagnosis (1) Vesico bullous or eczematous skin lesion on the face and exposed parts of the body, (2) Porphyrin estimation in fresh urine with *Ehrlich* aldehyde test—the urine shows red colour due to the formation of porphobilinogen, (3) Spectroscopic

examination of the urine shows uroporphyrin spectrum, (4) Histopathology shows edema of the collagen fibers with an intraepidermal bulla

Differential diagnosis (1) Epidermolysis bullosum and (2) Drug rash

Prognosis Is never cured

Treatment Protection of the liver with methionine (Neo methidine) Sometimes splenectomy is advocated

MYXEDEMA

Definition Myxedema is a metabolic skin disease due to the deficiency of thyroid hormone and is characterized by non pitting edema of the skin which is dry, rough waxy and with slow body movements

Etiology It is caused by the lack of thyroid function especially after thyroidectomy Occurs after the age of 40 years and is generally seen in women

Signs and symptoms Skin has a waxy feel and is dry and rough with nonpitting edema Lips become swollen and everted Swelling of the dorsum of hands and on the pretibial regions *pretibial myxedema* is a rare complication Hair becomes dry, lustreless and falls from the scalp and eyebrows Baldness develops and there is absence of hairs on the lateral half of eye brows Nails become discoloured and are cracked Teeth become carious Pads of fat appear on the shoulders Expressionless face and slow movements are characteristics Anaemia is associated

Prognosis Good

Treatment, Thyroid extract gr ½ by mouth twice daily should be continued for a long time Cortisone therapy may be given with the restriction of table salt Hyaluronidase may be injected locally in pretibial myxaedema

DISORDER OF CARBOHYDRATE METABOLISM

Diabetes Mellitus

The skin disorders due to the disturbance in the carbohydrate metabolism are found in diabetes mellitus The skin lesions are (1) Diabetic pruritus this may be localised or generalised In hyperglycaemia the itching is particularly localised to the perineal and genital regions, (2) Diabetic yellowness is due to the excess of carotene in the serum of a diabetic giving rise to the carotene colour of the skin A diabetic cannot properly convert the carotene into Vitamin A, (3) Diabetic gangrene—this occurs after some injury to the toes or fingers in a diabetic, (4) Trophic ulcer—this is an indolent, punched out and painless ulcer on the planter surface of the sole in a diabetic, (5) Necrobiosis lipoidica diabeticorum—is a sharply circumscribed reddish lesion with pigmentation at the periphery in a frank or latent diabetic Commonly seen on the legs and is found mostly amongst women, (6) Fungus infection—is very common in diabetics, particularly moniliasis gets a firm foothold in a diabetic Moniliasis vulva, intertrigo, paronychia are common

Diagnosis (1) Typical lesions, (2) Examination of urine for sugar, (3) Blood sugar estimation (4) Blood Vitamin A and C estimation

Differential diagnosis (1) Trophic ulcer due to leprosy and (2) Xanthoma tuberosum

Prognosis Is fair

Treatment Prophylaxis is to cut down carbohydrate intake and avoid over weight Curative is to have local treatment for fungus, trophic ulcer and pruritus together with anti diabetic regime Sometimes insulin therapy is advocated Sovental Jelly (Knoll) locally helps in pruritus

XANTHOMA

Definition Is a skin disease characterized by papular yellowish plaques of various sizes all over the body

Types (1) Xanthoma palpebrum or Xanthelasma, (2) Xanthoma tuberosum, (3) Xanthoma disseminatum and (4) Xanthoma diabeticorum

Etiology Xanthometosis is due to the disturbance in the lipoid metabolism in which the liver is also concerned Hyperlipemia is associated with Xanthomatosis cutis Vitamins are also responsible for its causation

People of any age may be affected but Xanthoma palpebrum is usually found after the age of 40 Both sexes may be affected

Signs and symptoms Xanthoma palpebrum is the most common type and is found on the medial and often upper or lower eye lid of one or both eyes and is also kown as Xanthelasma (Fig No 196) The lesions are oval yellowish plaques of about ¼ to ½ inch in length The narrow end is towards the inner or outer canthus of the eyes It does not itch It is only a cosmetic dis

figurement Xanthoma tuberosum may be a half pea-sized papule or plaque of 1 inch or 2 inches square or may be nodular in shape Lesions are yellowish in colour Commonly found on the joints and may be on the plams and soles The lesions of Xanthoma disseminatus



Fig No 196
Xanthelasma

are found on the flexure surfaces and in the axillae and mucous membranes There is no itching The lesions of Xanthoma diabeticorum are small yellowish papules half pea in size and are found on the extensor surfaces associated with diabetes Itching is present

Diagnosis (1) Yellow papules on the skin, (2) Blood sugar, blood cholesterol and Vitamin A are high, (3) Blood pressure is high, (4) Biopsy histo pathology shows typical Touton giant cells in the dermis Later on fibrosis replaces the foamy cells

Differential diagnosis Necrobiosis lipoidica diabeticorum

Prognosis Good in Xanthoma palpebrum but grave in other types

Treatment Diet should be fat free Insulin is valuable in Xanthoma diabeticorum Heparin in dose of

25,000 units twice a week for 2 weeks is also advocated Plastic surgery, improving liver function and Vitamin B_{12} therapy are advocated in Xanthelasma

PSEUDOXANTHOMA ELASTICUM

This is characterized by yellow papules on the flexure surfaces with cutis hyperplastica and without hyper cholesterolaemia Found in axillæ and sides of the neck Histopathology shows degeneration of the elastic tissues in the dermis No treatment is known

NECROBIOSIS LIPOIDICA DIABETICORUM

Definition Is a skin disease characterized by multiple oval yellowish plaques on the extremeties

Etiology Diabetes is associated but sometimes it may develop in a non diabetic also Disturbances of choles terol metabolism is said to be responsible Affects all

Sings and symptoms Lesion starts after some trauma and are oval, yellowish plaques of the size of the palm Affects legs thighs and forearms and is non itching

Diagnosis (1) Typical lesion with pigmented peri phery and yellowish centre, (2) History of trauma, (3) History of diabetes (4) Blood sugar, blood cholesteral and blood vitamin A are high, (5) Histo pathology shows homogenization of the collagen fibers in the dermis with necrobiotic change Blood vessels are obliterated

Prognosis Good

Treatment Insulin injectioned X'ray therapy, fat free and low carbohydrate diet is given

ACANTHOSIS NIGRICANS

Definition Is a skin disease characterized by pigmented papules in the axillae, groin, face and eyelids

Etiology Sometimes seen in tropical countries Is associated in elderly patients with malignant disease of the viscera which is the adult type or *malignant type* whereas in the younger people intestinal tuberculosis followed by direct *invasion of the adrenals may be the* cause which is the Juvenile type or *benign type* Theories *for the causation of acanthosis nigricans* are (1) Malignant disease of the gastrointestinal system *producing in patients an altered response to light,* (2) Involvement of the adrenal gland or the sympathetic *nervous system,* (3) *Disturbance of the endocrine glands* and (4) Vitamin C deficiency with hypervitaminosis A

Signs and symptoms Half pea sized black pigmented soft nodules may be found in the axillae, groins, perineum, genitalia and on the face May be found on the mucous membrane such as tongue and vagina Skin may be ichthyotic Alopecia may be associated Nail changes may be present

Diagnosis (1) Soft, pigmented nodules situated in the axillae, groin, face, (2) Ichthotic skin, alopecia with nail changes, (3) Biopsy—histopathology of the nodule shows hyperkeratosis with acanthosis and the stratum basalis shows excessive melanin formation

Differential diagnosis Keratosis follicularis, Ichthyosis

Prognosis Adult type is grave

Treatment No treatment is known In the juvenile type thyroid extract gr ¼ daily with high Vitamin A and in the adult type injection of suprarenal extract ACTH injection in both the types may be tried

CHAPTER XVI

DERMATOSIS DUE TO AVITAMINOSIS

"Intelligent treatment by diet is the greatest weapon available to preventive medicine" The greatest achievements in our knowledge is the research in the field of protein and the synthesis of vitamins

With the progress of civilization and to adjust with the changing customs of different races there has been a revolution in the diets throughout the ages The availability or dearth of food in the tropics has a tremandous effect on the nutrition The essential elements in the body metabolism are the proteins, minerals and vitamins With the discovery of vitamins and the establishment of its correlation with diseases it has been possible now to treat various skin diseases only with vitamin therapy It has been recognised to day that the vitamins are assential to maintain health and to act as a prophylaxis against various diseases

VITAMIN A

Etiology Due to unbalanced diet avitaminosis A is quite common in the tropics Vitamin A is formed in the gut wall and is stored in the liver Deficiency may be due to (1) deficient or unbalanced diet (2) to inefficient absorption of provitamin from the intestinal tract, (3) mineral oil intake (4) deficiency of vitamin E intake, (5) infectious diseases (6) parasites in the small intestines adversely affect the metabolism of vitamin A (7) choline is essential for storage of vitamin A, (8) hormones

are also responsible Age—all ages are affected, Sex—both sexes are affected equally in the tropics

Signs and symptoms When the deficiency is very mild the skin is dry and on scratching fine exfoliation occurs which looks like a white line such a mild condition is called *Xeroderma* which is more aggravated in winter When the deficiency is moderate and chronic the skin becomes dry, black and cracks appear on the skin like fish scale This condition is called *Ichthyosis* (Fig No 10 & 11) when localised hypertrophy or warty growths appear on the ichthyotic skin it is called *Ichthyosis hystrix* When the condition is much advanced and the patient dies soon after birth is called *Herlequin baby* Where the skin is like a crocodile and breathing is not possible There may be hyperkeratosis of palm and sole which is called *Hyperkeratosis plantaris et palmaris* When the vitamin A deficiency is chronic there is also besides dryness ofthe skin keratotic plugs appearing at the mouths of the hair follicles, sometimes with the loss of hairs, which are felt like thorns when it is called *keratosis follicularis* Skin looks stippled and gets exaggerated during winter Traumatic areas are particularly affected such as round the elbows, buttocks, knees, extensor surfaces of the extremities and sometimes on the body When this condition is further exaggerated the lesions conglomerate together and affect the face neck, chest, back, extremities and the skin becomes pigmented This condition is known as *Darrier's disease* Sometimes from keratosis follicularis stage the skin lesions become nodular, half pea sized in shape and get distributed round the elbows, buttocks and knees This condition is called *Phrynoderma* When the vitamin A

deficiency becomes very chronic the patient develops red pin head sized papular lesions on the skin in patches on the sides of neck, trunk, extremities, back of fingers alternating with normal skin accompanied with edema of face, body, hyperkeratosis plantaris and palmaris, there is also scaliness on the scalp with alopecia with dis coloured and cracked nails The skin lesions are sym metrical in distribution After several months the skin of the whole body becomes yellowish red in colour and somewhat atrophic Itching is the only symptom This condition is known as *Pityriasis rubra pilaris* Together with the skin lesion there may be Bitot's spots in the eyes, photophobia and xerophthalmia

Diagnosis (1) Typical dry skin alone in xeroderma and with fish scale like lesions or warty lesions in ichthyosis with pigmentation and follicular hyper keratosis in keratosis follicularis and Darrier's disease, half pea sized nodules in phrynoderma, with patchy, red papular lesions alternating with normal skin accom panied with hyperkeratosis palm and sole in pityriasis rubra pilaris, (2) Blood Vitamin A and C estimations show low blood levels, (3) Biopsy—histopathology shows in ichthyosis there is atrophy of the sweat and sebaceous glands with thinning of the epidermis, in keratosis follicu laris keratotic plug in the mouth of the hair follicle with hyperkeratosis of the epidermis and in Darrier s disease in addition there are *corps ronds* in the stratum granulosum and increased pigmentation in the stratum basalis In pityriasis rubra pilaris there is hyperkeratosis and parakeratosis of the stratum corneum, keratotic plugs in the mouths of the hair follicles, degeneration of the stratum basalis and perifollicular infiltration in dermis

Differential diagnosis (1) Lichen planus, (2) Exfoliative dermatitis and (3) Acne vulgaris

Prognosis Good

Treatment Prophylactic dose of vitamin A is 6000 i u daily A balanced diet should consist of meat, fish, egg, vegetables, milk, butter and ghee

Curative is to give by mouth Vitamin A in dose of 100,000 i u daily for 9 months Sometimes multiple deficiency is present and multivitamin is advocated (Panlyn of Calcutta Chemical) Protein diet is helpful Locally lotiocalamine or Soventol Jelly (Knoll) are helpful

VITAMIN-B COMPLEX

Vitamin-B deficiency causes edema of the skin which is called beri beri

Treated with high dose of vitamin B parenterally and with protein diet

Vitamin B_2 deficiency causes ariboflavinosis Signs and symptoms angular stomatitis, inflammation of both the lips called cheilosis, dermatitis in the nasolabial folds called dyssebacia with squamous lesions over the whole of the face Tongue has typical magenta colour with atrophy of papillae There is often photophobia and conjunctivitis The genitalia presents an erythematosquamous lesion which is very itchy All these lesions together go to form the *oro genital syndrome* There may be erythemato papular lesions on the nose with ulceration of the cornea

Treatment Daily requirement is 3 mg per day for an adult Riboflavine by injection or by mouth is

indicated High protein diet is helpful particularly liver, egg, milk Sovental jelly (knoll) locally helps

NIACINE

Nicotinic acid amide (niacine) deficiency causes Pellagra

Definition Pellagra is characterized by erythemato squamous skin lesions with gastrointestinal troubles and irritability

Etiology This condition develops in the tropics in adults mostly but children are no exception Both the sexes are equally affected

Niacine deficiency may be due to (1) Unbalanced diet, (2) Inhibition of synthesis in the gut caused during sulpha antibiotic therapy, (3) Intestinal infection such as giardiasis amaebiasis, blantidiasis and ascariasis

Signs and symptoms The disease has a prodromal stage characterized by dyspepsia, insomnia and erythematous lesions on the neck hands, and legs for 2 to 3 years Gradually the early erythematous skin lesions change to dry erythemato bullous further changing to papulo squamous with pigmentation (Fig No 197) Casal s necklace is the erythematous lesion round the neck becoming pigmented in a pellagrin The skin lesions are symmetrically distributed on the dorsum of both hands (Fig No 198) and to a variable length over the forearms dorsum of feet and the legs The face, neck and the part of the back become erythematous changing to blackish colour in the tropics The skin lesions on the face occurs on both the malor regions and on the nose like butterfly Mucous membranes look bright



Fig No 197
Pellagra



Fig No 198
Pellagra

red in colour Salivation is profuse Muscular weakness develops and burning sensation is felt in the mouth Sometimes ulceration in the angles of the mouth may be found In the late stage the skin becomes atrophic and pigmented The patient may have diarrhoea or constipation but sometimes alternate constipation and diarrhoea may be seen Dyspepsia becomes chronic Sprue or para sprue like symptoms may be found Insomnia, irritability, neurasthenia are commonly seen The patient sometimes develops psychosis Typical cases are rare Subclinical cases of pellagra are common in the tropics

Diagnosis (1) Symmetrically distributed, erythe mato squamous or papulo pigmented skin lesions on the

dorsum of the hands feet and round the neck with dyspepsia and irritability with insomnia in a patient, (2) Increased porphyrin in urine (3) Biopsy histopathology shows hyperkeratosis with parakeratosis in the stratum corneum acanthosis of the stratum mucosum, demarcation of the stratum basalis with increased formation of melanin In the dermis there is edema and dilatation of vessels

Differential diagnosis (1) Eczema, (2) Leprosy, (3) Syphilitic cutis and (4) Dermal leishmaniasis

Prognosis Is good in mild but grave in Acute cases

Treatment The whole vitamin B complex is given daily by injection and Niacin orally 100 mg Crude liver extract is injected intramuscularly 2c c every day The diet should consist of milk, protein (meat, fish and egg) vegetables (tomato peas) and fruits (mango, orange) and as a supplement multivitamin tablets Locally liniment Calamine is applied several times a day Exposure to sunlight is avoided

PINK DISEASE

Definition Is a skin disease of children characterised by redness of hands and feet with photophobia

Etiology Generally affects children from the age of three months Cause is not known Mercurial poisoning or vitamin B complex deficiency may be responsible

Signs and symptoms The child becomes restless, irritable and sleepless Anorexia is present with salivation Photophobia is marked After about a month the hands and feet become red and cold Itching is promi



Fig No 197
Pellagra



Fig No 198
Pellagra

red in colour Salivation is profuse Muscular weakness develops and burning sensation is felt in the mouth Sometimes ulceration in the angles of the mouth may be found In the late stage the skin becomes atrophic and pigmented The patient may have diarrhoea or constipation but sometimes alternate constipation and diarrhoea may be seen Dyspepsia becomes chronic Sprue or para sprue like symptoms may be found Insomnia, irritability, neurasthenia are commonly seen The patient sometimes develops psychosis Typical cases are rare Subclinical cases of pellagra are common in the tropics

Diagnosis (1) Symmetrically distributed, erythemato squamous or papulo pigmented skin lesions on the



Fig No 199
Kwashiorkor
(Ca e Dr D B Jelliffe)

Diagnosis (1) Weaning age, (2) Diarrhoea, (3) Irritability and (4) Skin rash

Prognosis Good when treated

Treatment Prophylaxis consists of good antinatal care of mother If the mother is nursing the child as is the common custom in the tropics the diet during lactation of the mother should be scientifically balanced

Curative consists in giving repeated blood transfusion to the child orally vitamin A in dose of (10 000 i u) in divided doses with Multivitamin solution by mouth Locally Liniment calamine should be applied 8 to 10 times during the day and night When the lesions are dry 1 p c Ung Ichthyol is applied

Diet is very important The child should be given normal diet consisting of rice, bread, dal (pulses), vegetable soup, meat and liver soup, fish, egg, and milk

nent which causes *secondary infection* *Sometimes* an erythemato papular rash is found on the body

Diagnosis Restless, irritable child with excessive salivation and red hands and feet

Differential diagnosis Eczema, Congenital syphilis

Prognosis Takes several months to get well Death sometimes takes place due to intercurrent diseases

Treatment Sedative is given such as syrup chloral hydrate 3 to 4 times a day for several days *Protein deit* Orally vitamin B complex and locally Sovental jelly

KWASHIORKOR

Definition Kwashiorkor is a nutritional skin disease of children charactrized by edema of hands and feet with pigmentation, *depigmentation*, *hepatomegaly*, diarrhoea and mental changes with stunted growth

Etiology Is not very common in the tropics as breast feeding is continued for about 2 years Found in India Age—below one year Sex—both sexes suffer It is a condition of malignant malneutrition

Signs and symptoms Edema is the earliest sign which appears on hands and feet Hairs are fine, rough and look reddish Macular errythematous rash appears on buttocks, perineum, groins, hands and fect and exfoliation with raw, red areas are often seen in infants with kwoshiorkar (*Fig No 199*) *Pigmentation* and hypopigmentation, microcytic anaemia, enlarged liver and associated *with palpable spleen* Diarrhoea is always an associated early symptom Children become fretful, restless and irritable Growth becomes stunted

CHAPTER—XVII

PIGMENT ANOMALY OF THE SKIN

May be (1) Hyperpigmentation and (2) Hypopigmentation

Causes of hyperpigmentation are —

A Congenital such as (1) Tropical races, (2) Freckles, (3) Xeroderma pigmentosum (Fig No 161) and (4) Incontinentia pigmenti

B Physiological as in pregnancy

C Physical agents Burns

D Chemical agents Drugs and Chemicals,

E Infection (1) Pediculosis, (2) Fungus, (3) Syphilis, (4) Leprosy (5) Kala Azar, (6) Malaria

F Endocrine (1) Pregnancy, (2) Addison s disease and (3) Hyperthyroidism

G Nutritional (1) Pellagra (2) Melanoderma due to difficiency of Vitamin A and Vitamin C and excess of Vitamin D and (3) Malnutrition

H Dermatosis such as (a) Psoriasis (b) Lichen planus, (c) Pemphigus, (d) Senile Keratosis, (e) Acanthosis nigricans and (f) Melano carcinoma

I Unknown cause such as chloasma

Causes of hypopigmentation are (1) Albinism and (2) Leucoderma

INCONTINENTIA PIGMENT

Definition It is a hereditary pigmentary disease occuring in children sometimes after birth

VITAMIN-C DEFICIENCY

Causes scurvy Clinical scurvy in the tropics is rare due to the consumption of plenty of green vegetables but subclinical cases may be seen The scurvy is characterized by the loss of weight, bleeding from the gums and in the skin The skin lesions consist of petechial haemorrhage with perifollicular congestion Hematomas may also be found Skin becomes rough

Treatment consists of (1) rest in bed, (2) Vitamin C (500 mg) is injected intramuscularly twice daily for a week and then vitamin C (200 mg) to be taken by mouth every 6 hours for a week, then 100 mg 50 mg, 25 mg before stopping the vitamin C therapy Diet should consist of milk, meat, fish, liver, vegetables, fruits (orange, lime)

CHAPTER—XVII

PIGMENT ANOMALY OF THE SKIN

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Causes of hyperpigmentation are —

A Congenital such as (1) Tropical races, (2) Freckles, (3) Xeroderma pigmentosum (Fig No 161) and (4) Incontinentia pigmenti

B Physiological as in pregnancy

C Physical agents Burns

D Chemical agents Drugs and Chemicals,

E Infection (1) Pediculosis, (2) Fungus, (3) Syphilis, (4) Leprosy (5) Kala Azar, (6) Malaria

F Endocrine (1) Pregnancy, (2) Addison s disease, and (3) Hyperthyroidism

G Nutritional (1) Pellagra (2) Melanoderma due to difficiency of Vitamin A and Vitamin C and excess of Vitamin D and (3) Malnutrition

H Dermatosis such as (a) Psoriasis, (b) Lichen planus, (c) Pemphigus (d) Senile Keratosis, (e) Acanthosis nigricans and (f) Melano carcinoma

I Unknown cause such as chloasma

Causes of hypopigmentation are (1) Albinism and (2) Leucoderma

INCONTINENTIA PIGMENT

Definition It is a hereditary pigmentary disease occuring in children sometimes after birth

Etiology It is a rare disease in the tropics It is said to be common in girls Starts early in infancy This is also known as Bloch Sulzberger *syndrome* Cause is not known but may be due to intrauterine virus infection or hypervitaminosis A with hypovitaminosis C

Signs and symptoms The child is born with hydro *cephalus or with other defects* After a week from the birth the child usually develops an erythematous rash all over the body Sometimes the rash may be bullous The rash disappears in about a week's time leaving pigmentation all over the body This pigmentation may start anytime from birth upto the age of two years Pigmentation usually disappears near about puberty but often it is permanent The child will have some malformations either in the form of alopecia, thickening of *palm and sole and hydrocephalus In a grown up child* malformation of teeth or absence of teeth, occular defects, congenital heart disease, epilepsy and even paralysis may be associated The pigmentation is found from the head to the feet and is typical Macular pigmentation of various patterns are seen Typical lesions are bizarre arborization in distribution The usual colour is the slaty colour but shades of brown to black may be found It is not itchy Sometimes patients feel discomfort in summer *due to the poor function of the sweat glands* General health may be good

Diagnosis (1) *Typical bizarre pigmentation of* chocolate colour of the skin of trunk in a child since birth, (2) *Biopsy-histopathology shows thinning of the* epidermis with hyperkeratosis and acanthosis Large amount of melanin in the dermis and specially perivascular in distribution Pigment granules look dropped

in the dermis and may be found in chromatophores and is called pigment incontinence

Differential diagnosis (1) Drug rash (2) Naevus, (3) Angioma serpiginosum and (4) Melanoderma

Prognosis So far the skin pigmentation is concerned it is usually permanent Rarely the pigmentation fades

Treatment No treatment is of any use I M injections of Vitamin C for a long time together with cortisone therapy and a diet deficient in vitamin A is helpful in some

CHLOASMA

Definition Is a localized symmetrical hyper pigmentation on either side of the face of females

Etiology No cause is known but ovarian dysfunction or liver dysfunction are found Commonly seen in middle aged women in the tropics Varieties are (1) Symptomatic when associated with uterine troubles and (2) Secondary when due to pressure or irritation

Signs and symptoms Macular pigmented spot of the size of a small coin appears on the malor region of one or both sides of the face Gradually they enlarge in size and become almost circular with about an inch in diameter Sometimes a linear pigmented patch appears on the bridge of the nose It is nonitchy and noninflammatory Colour may be brownish or blackish

Differential diagnosis From fixed drug rash

Prognosis It is difficult to cure

Treatment Vitamin C (Redoxon 500 mg) may be injected with vitamin A (Arovit 100 000 i u) by mouth

Testosterone propionate therapy is sometimes helpful it is due to hypersecretion of folliculin

Locally (1) Banoquine may be applied and the fresh solution of 5 p c monobenzyl ether of hydroquinone should be used for 2 weeks locally A bleaching cream is advocated containing bismuth oxychloride

MELANODERMA

Definition Is a chronic skin disease characterized by macular bluish or blackish pigmented plaques all over the body

Etiology It is quite common in the tropics Found generally in adults who do not take balanced diet Common in adults of both sexes Intestinal infection is commonly associated

Signs and symptoms Starts as multiple perifollicular pigmented macular lesions which enlarge and coalesce to form large plaques The colour deepens and from bluish becomes black Extremities are usually first affected symmetrically then it appears on the trunk, face and even on the scalp The face may sometimes be affected first It is slightly itchy Patient is healthy and does his or her work normally Insomnia is sometimes complained of

Diagnosis (1) Macular pigmented plaques all over the body in adults, (2) Estimation of blood vitamin A shows much low value, (3) Vitamin C estimation also shows a low value, (4) Gastric analysis shows hypochlorhydria, (5) Biopsy—histopathology shows slight acanthosis with excessive deposition of melanin pigments in the dermis below the stratum basalis

Prognosis Is good

Treatment (1) Vitamin C (500 mg) is injected, (2) Multivitamin is given by mouth, (3) Locally Liniment Calamine (4) High protein diet helps

ALBINISM

Definition It is a skin disease with complete or incomplete absence of pigments in skin, hairs and eyes

Etiology It is also known as congenital leucoderma Cause is not known but there is congenital absence of pigment in the skin, hairs and eyes The condition sometimes is familial Common in females Age—since birth In the tropics albinos are not rare

Signs and symptoms albino (Fig No 200) when the absene of pigment is complete but partial albinism when the absence of pigments is incomplete (Fig No 201) The skin is white The hairs are thin and white Photophobia is present Skin malignancy may develop due to the effect of the actinic rays of the sun

Differential diagnosis Leucoderma

Prognosis Is never cured

Treatment No treatment is of any use Should avoid sun 10 p c Para Amino Benzoic Acid ointment is applied Para Amino Benzoic Acid may be given by mouth in dose of 25 mg 4 times daily

LEUCODERMA

Definition This is a skin disease characterized by acquired absence of pigmentation from areas of skin

Etiology Classification (a) Idiopathic and (b) Secondary Common in the tropics Cause is not



Fig No 200
Albinism with
multiple carcinoma
(Case of
Captain S N Roy)



Fig No 201
Partial albinism

known in the idiopathic type but has been found to be associated with gastro intestinal derangement such as gastritis peptic ulcer amoebiases giardiasis, blantidiasis intestinal helminthiasis, intestinal tuberculosis, sprue para sprue and such other conditions which hamper the absorption of nutrition from the intestine Secondary type is due to local use of substances which inhibit melanin formation in the melanophore cells such as burns or chemicals and due to some skin diseases Seen in all ages except infants and in both sexes

Signs and symptoms Macular depigmented areas may occur anywhere on the body (Fig No 202 & 203) Usually starts as pin head spots on the extremities



Fig No 202
Leucoderma
(Girl aged 7 years)

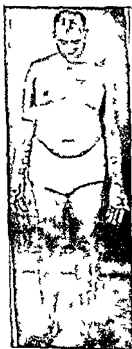


Fig No 203
Leucoderma

which coalesce and form big plaques The periphery is hyperpigmented May develop on vaccination scars (Fig No 204) Sometimes there is a red or black mole in the centre of a small oval leucodermic patch

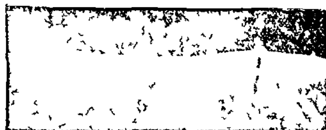


Fig No 204
Leucoderma of left forearm
(Post vaccination in a girl aged 9 years)

which is called *leucoderma acquisition centrifugum* Due to the absence of pigmentation the skin becomes sensitive to the sun s rays Depigmented spots may start either on the extremities or in the axillæ (Fig No 205) Itching may be present



Fig No 205
Leucoderma of axillae
(Case of Dr S C Mitra)

Diagnosis (1) Macular white plaque with pigmented periphery appearing anytime in life after infancy, (2) cause is always present either the use of rubber gloves badly tanned shoes, cosmetics, nickel watch band or plastic watch band (3) Biposy histopathology shows absence of melanin in the stratum basalis

Differential diagonsis (1) Tinea versicolor, (2) Seborrhoea (3) Dermal Leishmaniasis (depigmented stage) and (4) Albinism

Prognosis Is fair Some get well others change colour and take a long time to develop pigmentation There are still some which cannot be cured

Treatment Prophylaxis is to avoid exposure to sun and examine stool for intestinal infection which when found should be treated properly

Curative is to examine stool and treat the parasitic infection Then a course of emetine hydrochlor gr 1 (one grain) per day is injected intramuscularly daily for 6 days The liver is improved by methionine and choline (such as Neomethidine Neo Pharma) by injection together with Vitamin B complex by mouth Locally an ointment is rubbed containing tar but on the mucous membranes a lotion is painted containing oil of Bergomot with 60 p c Spt Rectificatus

In the tropics it is being treated by an indigenous herbal product The Indian physicians (Vaids and Hakims) have been treating leucoderma for several thousand years with the powdered seeds of Bouchi This has been put in the market as Leudermol (Smith Stanistreet Calcutta) ointment and oil for local use and are being used with a great amount of success Recently from Egypt another herbal preparation has claimed pigment producing function and is now available as "Meladinine (Laboratory Grimault, Paris) lotion for application with good results

CHAPTER XVIII

DISEASES OF HAIR

CANITIS—Is the change in colour of the hair in a young person from black to white All or few hairs may be affected Cause may be (1) Psychosomatic (2) after acute illness, (3) on patches of alopecia areata and on leucoderma, (4) deficiency of vitamin B complex and vitamin A (5) in hyperpituitarism Treatment No treatment has any effect High vitamin A (Arovit and Para Amino Benzoic Acid by mouth may be given Hair may be dyed

LEUCOTRICHIA ANNULARIS—Is a rare anomaly of the hair characterized by the presence of white bands on the shaft of the hair This is due to the presence of bubbles of air in the hair shaft

FRAGILITAS CANINUM is a condition in which the hair splits either longitudinally or transversely

TRICHORRHEXIS NODOSA is characterized by incomplete *multiple transverse fractures of the shaft of* tha hair which look like nodes

MONILETHRIX is characterized by multiple beads on the shaft of the hair

HYPERTRICHOSIS

Definition Is the abnormal growth of hair on different parts of the body It is also known as hirsutism

Etiology Two types are known (1) Congenital type as *in nevus* pilosum and in dog faced persons

where long hairs are seen all over the body including the face except on palm and sole (2) Acquired type as in pregnancy Cushing s syndrome (3) Local use of androgen (4) In women androgen produces growth of hair, (5) In virilism

Signs and symptoms Partial congenital type is seen in nevus pilosus Localized hypertrichosis is commonly seen on the sacral region in case of spina bifida (Fig No 206) Excessive growth of hairs may occur on face of women Commonly seen in women



Fig No 206
Hypertrichosis Tuft of hairs on the sacral region of a child aged 2 years

after menopause It produces a psychological upset Investigation in young woman with hirsutism are (1) 17 Ketosteroid high in urine, (2) Skiagram of sella tarcica-abnormal (3) Pyelogram, (4) Abnormal carbohydrate tolerance test

Treatment Depends on the cause When due to pituitary tumour X ray therapy is helpful Operation is advised in case of suprarenal tumour Epilation

by electrolysis of the hairs is helpful The hairs may be bleached with the application of hydrogen peroxide

HYPOTRICHOSIS

Also known as alopecia or baldness

Varieties of alopecia (1) Idiopathic, (2) Secondary and (3) Psychic

(1) Alopecia idiopathica may be due to (a) when the hairs and hair follicles also are absent at birth together with defective dentition is known as *congenital ectodermal defect*, (b) Baldness which starts from the frontal regions and progresses backwards in young adults is called *lopecia prematura* Alopecia generally develops in the old age due to the changes in the gonads and is known as *Alopecia senilis*

(2) Secondary alopecia may be due to (a) Hair dressing, (b) Massage of scalp, (c) Fungal, (d) due to burns, (e) due to skin diseases like scleroderma, lupus erythematosus, lupus vulgaris, syphilis (Fig No 207)



Fig No 207
Syphilitic alopecia

(f) due to seborrhera, (g) due to coccal infection of the hair follicles causing baldness of various shapes

tinue to beat in sinus rhythm. Thus, it is probable that the R wave of the seventh extrasystole and the iso electric line between the ninth and tenth include a P wave.

The form of the QRS complex varies in each individual case according to the point of origin of the tachycardia and the spread of excitation occasioned by it. As with isolated ventricular extrasystoles, extrasystoles of ventricular tachycardia, as a rule, are not conducted back to the auricle, so that in ventricular tachycardia actually an auriculoventricular block is often present. The auricles contract regularly in a normal way under the control of the sinus node; yet one can find P waves only at a few places, since, being small, they are lost in the QRS complexes and T waves of the extrasystoles.

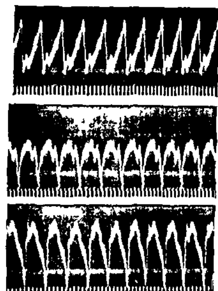


FIG. 189. Paroxysmal ventricular tachycardia.

The ventricular rate in paroxysmal ventricular tachycardia varies. It may exceed the existing normal rhythm only a little, but there are also minute rates of 250 and more. In such cases ventricular flutter is very often said to be present (see p. 295). Ventricular tachycardias are usually regular. Figs 188 and 189 show the great regularity of typical cases. It is often asserted that they show irregularities which are demonstrable by auscultation (Strong and Levine), but this is usually impossible because the fast rate prevents marked differences in the length of diastole, and more recent investigations have failed to corroborate this assertion (MacKinnon). Irregularity occurs only in some special types of ventricular tachycardia (Fig. 191). But changes in the intraventricular conduction (Fig. 190) and occasional summation with auricular contractions may change the intensity of the heart sounds.

Fig. 189 shows short sections of a tracing in all leads from a case of paroxysmal ventricular tachycardia. Again abnormal ventricular complexes in very rapid sequence are seen. The minute rate amounts to 300. The diagnosis of ventricular tachycardia is substantiated by observation after the end of the attack, when an entirely normal electrocardiogram interrupted by single ventricular extrasystoles

was found these extrasystoles exhibit the same appearance as those which compose the tachycardia

Just as in rare cases *single* extrasystoles may develop above the bifurcation of the bundle and then spread normally in the ventricle so at times in a ventricular tachycardia with a similar high origin of the stimulus the QRS complexes may exhibit a normal form The distinction between auricular tachycardia and atrioventricular tachycardia is then difficult (see p 438) On the other hand occasionally the presence of *ventricular* tachycardia may be wrongly diagnosed since the ventricular beats in an auricular tachycardia may appear abnormal owing to fatigue of the conduction path and aberrant conduction in the ventricle the development of an auri

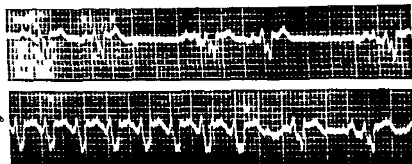


FIG 190 Case of disturbance of intraventricular conduction (a) auricular bigeminy and (b) end of an attack of paroxysmal auricular tachycardia

cular tachycardia in a patient with disturbance of intraventricular conduction may also cause confusion

Fig 190 shows auricular bigeminy The tracing was obtained from a patient with a mitral valve lesion and myopericarditis The P waves of the normal beats are widened and bifid An auricular extrasystole appears after each normal beat In the lower tracing (Fig 190b) the spontaneous end of a paroxysmal auricular tachycardia is seen Since the auricular extrasystoles then followed each other so rapidly the P waves are no longer distinct If the electrocardiogram had been taken only in the attack one would have been inclined to assume the presence of a ventricular tachycardia because of the rapid sequence of the abnormal ventricular complexes

In discussing ventricular extrasystoles and bigeminies the important fact was emphasized that variation in form of the extrasystoles and a constant change in their appearance favour the diagnosis of myocardial disease often it is not only the myocardial


Fig 191 Paroxysmal ventricular tachycardia (after digitalis) with changing in part alternating forms of extrasystoles

lesion, but the digitalis which in a diseased heart brings about this kind of extrasystoles In some cases (through the continuation of digitalis therapy), these extrasystoles multiply, so that one finds a chain of extrasystoles with constantly changing appearance At times one also sees an alternation of two forms These tachycardias are also called terminal tachycardias (Gallavardin), since they often appear just before death If the tachycardia has been produced by digitalis therapy it may vanish with the timely interruption of therapy and by the administration of quinidine

In Fig 191 there is a typical example of a ventricular tachycardia of this kind It appeared in a severely decompensated patient with a coronary lesion, who had been treated with small doses of digitalis (3 × 0 05 gm daily) Initially the ventricular complexes are similar in shape, but in contrast to other ventricular tachycardias they appear at irregular intervals After the fifth extrasystole ventricular complexes of various shapes are seen, and soon the two forms of extrasystoles continually alternate with each other

The alternation may be absolutely regular for hours and days Occasionally there is also *an alternation of a longer and a* shorter diastole but frequently the rhythm is very regular If the rhythm and form of the ventricular contractions change continuously the condition is called "anarchie ventriculaire" (Clerc and Levy)

The alternation is best explained by disturbances of intraventricular conduction (Scherf and Kisch)

Paroxysmal Auricular Tachycardia

Paroxysmal auricular tachycardias are composed of a chain of auricular extrasystoles As with single extrasystoles the appearance

of the P wave may vary Inverted P waves are very common but they may be positive notched, or broadened There are tachycardias which are only a little faster than the sinus rhythm of the case concerned and there are tachycardias with very fast rates As in flutter disturbances of conduction between the auricle and ventricle may appear in fast auricular tachycardias, so that as a result of fatigue of the conduction system the beats are conducted aberrantly to the ventricle in such cases wide abnormal QRS complexes appear These tachycardias may then give the impression

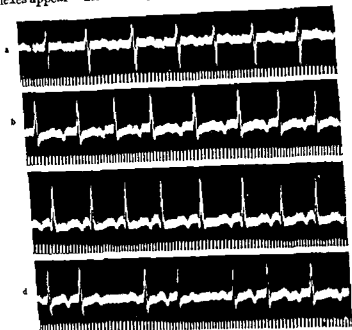


Fig 192 (a–c) Paroxysmal auricular tachycardia (d) An auricular bigeminy and beginning of a new attack

that they are ventricular in origin As with single extrasystoles one must always try to determine whether or not the ventricular beats are preceded by P waves

In Fig 192 the three upper tracings are the three standard leads from a case of paroxysmal auricular tachycardia The rate amounts to only 127 In this case it happens that there are inverted P waves in all leads which are immediately apparent after the T waves of the preceding beat In Fig 192d one notices a single auricular extrasystole after each of the first two normal beats Following the third normal contraction a new attack of tachycardia begins The

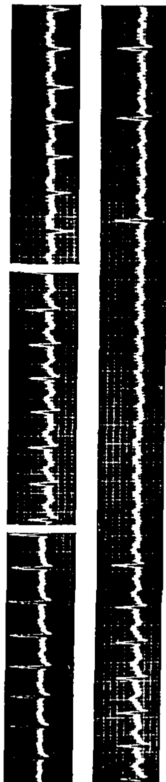


Fig 196 Upper tracing shows auricular flutter with 2 1 block in all leads Differentiation with certainty from auricular tachycardia was possible only by means of carotid pressure test (lower tracing)

duced by the inhalation of amyl nitrite), Fig 195b (unquestionable paroxysmal auricular tachycardia), and Fig 195c and d (auricular flutter with 2 1 block) may resemble each *other so closely as to* be confusing In all three types of tracings we see slender normally conducted ventricular complexes in which a P wave or a flutter wave may, but *need not*, be concealed

In very rare cases, auriculo ventricular tachycardia (from the middle of the node) or ventricular tachycardia (above the bifurcation of the bundle of His) may show the same picture

Sinus tachycardia is excluded immediately with relative ease when the sudden beginning or the abrupt end of an attack is recorded or observed this never happens in sinus tachycardia If the patient *presents a continuous fully developed* tachycardia a test should be made to determine whether an alteration of position of the body or slight exertion (repeated sitting up and lying down in bed) causes a slight acceleration of the pulse (This test as stated earlier never fails in sinus tachycardia) In paroxysmal auricular tachycardia or in auricular flutter change of position of the body or slight exertion does not influence the rate It is therefore apparent that the diagnosis cannot *always be made from the electro* cardiogram alone rather a functional test is necessary for this purpose Only in rare cases of paroxysmal auricular or ventricular tachycardia does the heart rate increase following physical effort (Scherf and Weissberg)

If *no* acceleration appears after slight effort then auricular flutter with 2 1 block or paroxysmal auricular tachycardia is present Occasionally one may produce a doubling of the cardiac rate through increased effort, and thus prove the presence of flutter with certainty (p 310) In other cases pressure on the carotid sinus provides evidence for differentiation In sinus tachycardia this pressure is often without effect or produces only a transient slowing which is maintained only for the duration of pressure Paroxysmal auricular tachycardia may cease during carotid pressure but slight and transient slowing does not occur (p 347) In auricular flutter the flutter waves may become visible through the disturbance of auriculoventricular conduction since the ventricular complexes which concealed the F waves may disappear during the exertion of pressure

In Fig 196 a regular tachycardia is reproduced at first glance one cannot say whether it is an instance of sinus tachycardia paroxysmal auricular tachycardia or auricular flutter Sinus tachycardia could easily be excluded since the rate was unchanged after exercise Auriculoventricular conduction was depressed by means of pressure on the left carotid sinus with the result that distinct flutter waves appeared (lower curve)

But there are cases in which carotid pressure *and* exercise do not change the tracing then a distinction between auricular tachycardia and flutter cannot be made We shall see later that the therapy for these two conditions is identical so that an inability to distinguish them is not of therapeutic importance

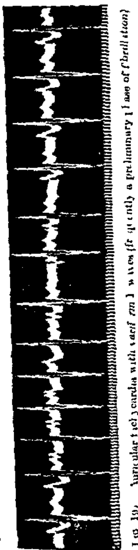


Fig 197 Auricular tachycardia with variform P waves (frequently a preliminary phase of fibrillation)

Just as *different forms* of QRS complexes sometimes appear in ventricular tachycardias a peculiar type of *auricular tachycardia* exists in which the shape of the P waves varies At the same time an arrhythmia is also present Thus we see in Fig 197 *constant*

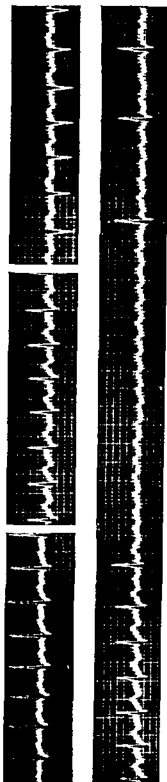


Fig 196 Upper tracing shows auricular flutter with 2 1 block in all leads Differentiation with certainty from auricular tachycardia was possible only by means of carotid pressure test (*lower tracing*)

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of the veins of the neck but frequently the venous pulse curve must be recorded

Fig 199 is reproduced from a case of paroxysmal auricular flutter in which entirely abnormal ventricular complexes temporarily appear through *fatigue of a part* of the intraventricular conduction system (the minute rate amounts to at least 300) with the result that one might assume a ventricular tachycardia was present *The spontaneous transition* of the normal ventricular complexes into abnormal ones may be seen in the record Since the irritability of the muscle fibres continually changes intraventricular conduction in such cases is at times normal at times abnormal All the time the tachycardia due to the abnormal stimulus formation in the auricle goes on (The tracing was obtained from a child five years of age)



Fig 199 (Lead II) Paroxysmal tachycardia (probably auricular flutter with 1 block) and (in second part of tracing) disturbance of intraventricular conduction

Clinical Aspects

Paroxysmal auricular and ventricular tachycardias are composed of auricular and ventricular extrasystoles For this reason *in discussion of the clinical aspects and therapy many points should* be repeated which have been mentioned earlier in the chapter on extrasystoles

The tachycardias vary considerably in their manner of appearance and duration They occur at all ages Often the tachycardias have been compared to epilepsy In both states an attack may occur once in a lifetime but it is also possible for them to recur daily Epilepsy as well as tachycardia may be the result of an organic disease but both states may also be idiopathic that is they may appear without any ascertainable cause

In the tachycardias *first one should always attempt to determine* whether an auricular or ventricular tachycardia exists The auricular tachycardias are much more common than the ventricular they also have a better prognosis If in a case of auricular tachycardia the underlying pathologic condition progresses auricular fibrillation may develop this is not dangerous and represents a

variation of the diastole and changing form of the P waves More over, the P–R interval changes continually and certainly in connection with the length of the preceding diastole Sooner or later these tachycardias often are transformed into auricular fibrillation, just as the corresponding ventricular tachycardias (Fig 191) may pass into ventricular fibrillation

In Fig 198 short sections are reproduced of the electrocardiograms of two cases studied only during the tachycardia As long as one is without knowledge of the electrocardiogram outside of the

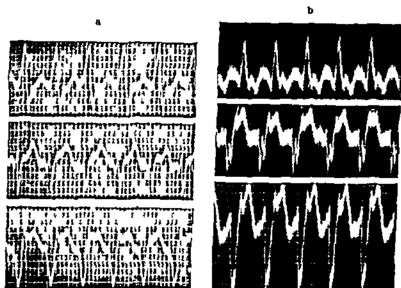


FIG 198 Two cases of paroxysmal tachycardia From these electrocardiograms it is impossible to determine what type is present

attack, the diagnosis of the type of tachycardia present in these cases is impossible The condition might be auricular flutter or auricular tachycardia in which the ventricular complexes are abnormal through simultaneous myocardial disease (organic disturbance of intraventricular conduction) The myocardium may be normal and the abnormality of the ventricular complexes might be merely the result of fatigue of certain branches of the conduction system (functional disturbance of intraventricular conduction ' aberration) On the other hand ventricular tachycardia may be present In some cases one succeeds in recognizing a ventricular tachycardia by the venous pulse During ventricular tachycardia the auricles continue to beat normally in sinus rhythm In rare cases one recognizes the slow auricular rhythm by a mere inspection

constipation meteorism excitement in others without any ascertainable reason In one individual they last for minutes, in another for days even for weeks Each case exhibits different behaviour The appraisal is easy when the patient comes with a long history and can furnish information in respect to all details Under these circumstances by careful inquiry one may gain an idea of the frequency of the attacks and their duration in the future The situation is different however when the patient comes *during or after the first attack* In this instance the prognosis should be determined only with great care Usually one will prefer to defer the decision until a period of observation permits sound judgment

If the patient reports that attacks have existed for years and the examination reveals normal cardiac findings then one will as in extrasystoles judge the tachycardia as a harmless but annoying functional disturbance But if the patient is seen during or shortly after the first attack he must be observed for a time despite the negative outcome of the first examination the tachycardia may be the first and only sign of an organic myocardial disease

Thus paroxysmal ventricular tachycardia earlier than any other sign may indicate coronary sclerosis a chronic myocarditis or a diphtheritic myocardial lesion Since coronary thrombosis has become more common the ventricular tachycardias are a more frequent undesirable complication If one finds them with varying forms of the ventricular complexes then as mentioned earlier an organic disease must always be suspected A digitalis effect must also be considered However auricular tachycardias with P waves of varying forms are always the result of an organic disease and never the effect of digitalis Usually they become transformed into fibrillation

Most patients who suffer from paroxysmal tachycardia perceive the sudden lightning like beginning and the abrupt end of the attacks so distinctly that this form can by inquiry be differentiated from other types of palpitation To be sure there are also patients who give vague reports and despite the existence of a tachycardia do not mention palpitation but only its resultant manifestations Premonitory signs which anticipate the beginning of the attack are rare

Anginal pain is a very common accompaniment of paroxysmal tachycardia In the discussion of the consequences of tachycardia in fibrillation it was stated that a diminution of the minute volume to less than one half and a fall of blood pressure occur with rapid tachycardia Since with increased rate of contraction the heart

complication which is susceptible to treatment But if ventricular tachycardia is present, the appearance of fatal ventricular fibrillation is possible

In the ventricular tachycardias the ventricles contract without preceding auricular action Earlier reference (p 300) was made to the importance of auricular contraction particularly in tachycardias, for rapidly augmenting the ventricular content during diastole In other words ventricular tachycardias should be regarded less favourably than auricular tachycardias of the same rate

There are two groups of auricular tachycardias At times (with a low rate) the auricles may contract so early in diastole that they succeed in forcing some blood into the ventricles (see Fig 192) But with increasing rates the P waves merge more and more with the T waves, that is, the auricles contract when ventricular systole is still complete If the auricles contract when the ventricles are still in systolic contraction, the former cannot empty their contents into the ventricles Since the orifices of the great veins in the auricles are not protected by valves but are closed during auricular systole merely by a special muscular mechanism this obstruction is easily overcome, this readily leads to a reflux of blood into the veins The blood which has just returned to the heart from the veins is again ejected from the auricle back into the venous system so that the liver rapidly enlarges and stasis in the neck becomes extreme When the auricular systole is superimposed upon the *ventricular it is called "engrafted (Wenckebach) It is under*standable, therefore that the prognosis in tachycardias in which superimposition occurs is much more unfavourable than when it is absent Fig 184 shows clearly that above a certain rate the diastole becomes so short that superimposition is inevitable The rate at which this takes place is approximately 180 and this was called by Wenckebach the *critical rate* In tachycardias with lower rates *superimposition may exist, if for example conduction is prolonged* In auricular tachycardias with a rate of 180 or more, it *always* exists

In a tachycardia at first the type (auricular or ventricular) and then the rate must be investigated in auricular tachycardia the presence of auricular superimposition must be determined all these features are important

It is also essential *to determine the frequency and duration of* the attacks In one patient they recur at intervals of years, in another almost daily, in some only during or before the menstrual period or in pregnancy (as extrasystoles) or only with marked

constipation meteorism excitement, in others without any ascertainable reason In one individual they last for minutes in another for days even for weeks Each case exhibits different behaviour The appraisal is easy when the patient comes with a long history and can furnish information in respect to all details Under those circumstances by careful inquiry one may gain an idea of the frequency of the attacks and their duration in the future The situation is different however when the patient comes *during or after the first attack* In this instance the prognosis should be determined only with great care Usually one will prefer to defer the decision until a period of observation permits sound judgment

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muscle needs a great deal more oxygen, such increase of rate also leads to a relative diminution of cardiac blood supply when compared *to cardiac performance* This reduction of perfusion provokes pain which may show all transitions between slight burning and pressure to most severe angina pectoris (see p 213)

Confusion of the anginal pain of coronary thrombosis with the anginal pain of paroxysmal tachycardia is not rare Severe distress maintained for hours in the midst of complete well being and without visible cause appears in both states (p 214)

The reduction of the stroke volume and the marked lowering of blood pressure appearing at the onset of the attack provide the reason for vertigo and weakness The attacks of unconsciousness are considered in the section devoted to Stokes Adams attacks (p 387)

The stasis which rapidly occurs " behind " the heart (in the veins of the neck) and the resultant acute engorgement of the liver may result in *vomiting*, many patients complain especially about this event, and other symptoms may remain in the background

A peculiar symptom at present scarcely investigated which accompanies paroxysmal tachycardia is *urina spastica* The patient reports voiding large amounts of clear watery urine This flood of urine often comes soon after the beginning at times during the attack, and rarely after its cessation The presence of this symptom supports the diagnosis although it must be conceded that urina spastica occurs in other syndromes and in disturbances of the vegetative nervous system

If the auricle contracts simultaneously with the ventricle (for example in ventricular tachycardias) superimposed waves appear in the neck veins they may make themselves unpleasantly obvious to the patient as " throbbing "

Extrasystoles in Paroxysms

In the forms of paroxysmal tachycardia discussed up to the present there may exist an interval of variable duration between the individual attacks at times this period of normal cardiac rhythm or at most isolated extrasystoles may last for years But there is a variety of paroxysmal tachycardia which requires special mention It concerns cases in which the individual attacks are relatively short and last for only a few seconds or minutes One or two normal beats occur and then another attack of tachycardia begins This phenomenon is constantly repeated There may be scarcely two, rarely several normal beats in sequence without extra systoles appearing between them *One* short attack follows another

so that these attacks were designated by Gallavardin as *extrasystolie a paroxysmes tachycardiques* The same syndrome has been called repetitive tachycardia by Parkinson and Papp



Fig 200 Ventricular extrasystoles in paroxysms

Fig 200 shows these extrasystoles in paroxysms After two normal beats a series of four ventricular extrasystoles follows After two more normal beats a longer series of extrasystoles occurs

In other cases auricular extrasystoles with similar characteristics occur

In these patients the abnormal mechanism gives rise to multiple extrasystoles so constantly and stubbornly that even the first or second normal beat after the end of the tachycardia precipitates a new attack Every normal impulse acts as a new incentive scarcely has its action on the extrasystolic centre declined when it precipitates the same disturbance again It is like a clonus appearing after every stimulation of the triceps muscle

Patients with this variety of extrasystoles at first make a good impression since the individual attacks last for only a short time and long post extrasystolic pauses which are always found after them balance the disturbance But it soon becomes evident that these cases must be viewed less favourably than the ordinary tachycardias If with the latter we succeed in removing the attacks by measures to be described later (p 347) we may anticipate that freedom from the symptoms will persist until the next attack and as a matter of fact another attack may never occur

The situation is different in extrasystoles in paroxysms which are under consideration in this section Here also the administration of *large* doses of quinidine or digitalis is successful since the extrasystoles vanish and sinus rhythm returns But if the dose of the drug administered is reduced even by *a little the tachycardia* recurs Since large doses of digitalis or quinidine cannot be given over a long time without danger and since small doses are inadequate soon it is realized that all therapy is useless for any length of time and that it suppresses the abnormal mechanism of the heart only very transiently Medicinal

therapy is therefore ultimately abandoned and the future course of the patient varies There are cases in which these tachycardias slowly subside, often only after years The individual attacks become progressively shorter and proportionately more *normal* beats are inserted between them If this is not the case, however, the heart is gradually injured by the steady acceleration which is maintained for years The dilatation of the heart which appears sooner or later leads to relative mitral or tricuspid insufficiency, extreme stasis develops, and finally death occurs The pathologist finds healthy valves and no signs of organic muscle alteration (Scherf and Kisch)

Mechanism of Paroxysmal Tachycardia

According to the results of available investigations the *origin* of paroxysmal tachycardia is attributed in most cases to a very frequent formation of stimuli in the specific fibres of a stimulus forming centre, that is, to the same event that gives rise to extra systoles They always begin a definite interval after a normal beat like extrasystoles they are coupled

Paroxysmal tachycardias have also been ascribed repeatedly to a circus movement and until very recently this explanation has been supported in many modified forms but for most cases it certainly has no validity Before and after the tachycardia repeatedly single extrasystoles are seen which show all the characteristics of those which compose the tachycardia Tracings which resemble Fig 246 cannot be explained by circus movement since the long pauses between extrasystoles are impossible if they are caused by a circulating wave such a wave must move incessantly In animal experiments it is easy to produce ventricular tachycardias and to prove that they originate in a circumscribed centre For example, warming of this centre with a thermode accelerates the rate

The alternating tachycardias (see Fig 191) were frequently explained by an alternating action of two centres Recent investigations favour the existence of the formation of stimuli in *one* centre combined with disturbances of intraventricular conduction (Scherf and Kisch)

The application of solutions of sodium or barium chloride and aconitine to circumscribed areas of the surface of the ventricles causes not only extrasystoles but also paroxysmal tachycardias again proving their origin from a circumscribed area of the heart without a circus movement mechanism (Piccione and Scherf) Warming of the focus of origin accelerates these tachycardias

All transitions exist between single extrasystoles and paroxysmal tachycardia The difference is a matter of quantity and not of quality

Treatment of Paroxysmal Tachycardia

The management of paroxysmal tachycardia may be divided into the treatment of the attack itself and measures for the prevention of new attacks

If the patient is seen in an attack it is well not to have recourse to drugs at once but an attempt should be made to end the paroxysm by means of one of the following vagal reflexes

At first *carotid pressure* is tried Formerly it was called vagus pressure since the notion prevailed that the heart and the tachycardia could be depressed by pressure directly upon the cervical vagus and through direct excitation of the vagus But the experience of investigators has shown that pressure crushing or tearing of the vagal trunk exposed by an operation in the neck is entirely devoid of influence on the heart (Winterberg) Several cases have been reported in which light pressure on the neck, certainly insufficient to influence the vagus trunk which lies deep behind the vessels of the neck depressed the heart and it has been shown that pressure was also effective in patients in whom the vagus had been previously sectioned by operation upon the side receiving the pressure (Scherf) Finally Hering proved in 1923 that no direct excitation came under consideration in so called vagus pressure but rather a reflex which emerged from the site of bifurcation of the carotid artery

Below the division of the common carotid into the external and internal carotid arteries there is a distinct expansion of the vessel the carotid sinus At this place the sinus nerve arises it unites with the glossopharyngeal and runs centrally with it to the vagus centre The two vagi represent the centrifugal path Mechanical stimuli and pressure on the carotid sinus lead to depression of the heart and in a majority of instances cause attacks of paroxysmal tachycardia to vanish The pressure is applied in such a manner that it is exerted at the upper border of the thyroid cartilage upon the carotid artery (anterior to the sternocleidomastoid muscle) which is pressed toward the vertebra Since pressure may be followed by immediate cardiac standstill it is applied only with the patient in the recumbent position Moreover it is advisable to check the cardiac rate at the same time The degree of pressure must be adapted to the individual case In some patients very light contact with the skin suffices in others quite strong pressure is

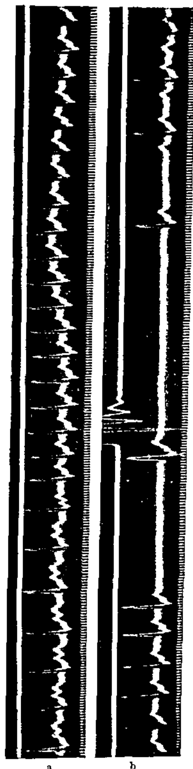


Fig 201 (a) Beginning of a paroxysmal auricular tachycardia (b) End of the attack (by carotid pressure)

necessary If *the first trial fails*, one may with advantage press somewhat higher or lower because the exact site of bifurcation must be compressed, pressure at any other place being ineffective Owing to anatomic variations, the site of bifurcation of the carotid artery is higher or lower in different people

In Fig 201a there is at first a regular sinus rhythm with a conduction time lengthened to 0 28 second The T wave of the seventh normal beat contains an auricular extrasystole which initiates a paroxysmal auricular tachycardia Fig 201b shows how the tachycardia is *abolished by carotid pressure (right sided)* As long as merely light pressure was exerted the tachycardia *continued but it stopped as soon as* the pressure was increased Since the pressure on the carotid sinus was continued for some time the heart ceased to beat for a moment after the cessation of the tachycardia Two auriculo ventricular beats (without P waves see p 398) appear, followed finally by the return of normal sinus rhythm After the long pause the conduction time is *shortened owing to better recovery* of the conduction system The cessation of carotid pressure is indicated on the white line above the electrocardiogram (stimulus signal)

Pressure is usually effective on the right side But there are cases in which it succeeds on the left side as well or on the left side *only* If the rules given are observed carotid

pressure is devoid of danger. It is obvious that pressure must not be exerted upon both sides simultaneously.

Many patients soon learn to exert the pressure *themselves* and can immediately abolish the attacks without medical assistance.

If carotid pressure is ineffective, a trial is made *with another* vagal reflex. There are patients who can abolish an attack by deep inspiration or by holding the breath; *others succeed through the* Valsalva experiment (the patient is advised to press down as in defæcation). Bulbar pressure *is very effective, especially* in young people. The patient looks downward and closes the eyes, and then pressure is exerted *with increasing force on* both eyeballs.

Sometimes it is possible to abolish the attack by having the patient *bend forward as far as possible*. This is supposed to increase

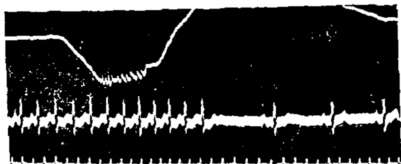


FIG. 202. Termination by deep inspiration of attack of paroxysmal auricular tachycardia.

spinal fluid pressure which in turn heightens vagal tonus. Deep *bending* causes a bradycardia in *the healthy individual*.

In Fig. 202 there exists a paroxysmal auricular tachycardia which could be *confused very easily with auricular flutter with full* conduction. Each attack was ended by deep inspiration (inspiration is shown in the respiratory *curve with the excursion upward*).

Finally there are patients who can immediately end a paroxysm by inserting a finger into the throat in order to produce vomiting. Often they learn this without instruction. In the course of their attacks acute hepatic stasis may occur and produce a sense of nausea and vomiting. The patients note once or twice that the attack ends with the initial retching. Subsequently they no longer wait until the vomiting comes spontaneously, but attempt to produce it immediately by inserting the finger into the throat, thus causing an emetic reflex.

In some patients one reflex is more effective, while in others it is

a different one , in a few minutes all are tested Only when they fail is *medicinal therapy* necessary Here quinidine should be mentioned first , it can be injected during the attack or may be given by mouth

An injection of quinidine may have an almost magical effect (Hecht and Zweig, Singer and Winterberg) The result appears so rapidly that at times the attack ceases before the injection is completed Nevertheless, great caution is necessary, since collapse may be observed after the intravenous injection of quinidine

Physicians who dwell in malarial regions and who inject large amounts of quinine are aware that now and then sudden death occurs after an injection If, for example, 0 2 gm (3 grs) of quinine or quinidine is given to a healthy dog, only very slight effects are seen But the same amount in another animal, with the experiment continued for a longer time, with loss of blood or a damaged heart, may result in diastolic cardiac standstill If a patient seeks relief in an attack and the status of his heart outside of the paroxysm is unknown, an injurious effect of the injection is quite possible Since in an attack one simply hears a pendulum rhythm of the sounds which rapidly follow one another, and since murmurs or other abnormal auscultatory phenomena cannot be heard, a profound alteration of the myocardium or a marked valvular lesion may thus not be recognized Owing to the small stroke volume and the low blood pressure during the attack the second sound is often inaudible

We should also realize that myocardial injury may occur during the attack and may be caused by it in otherwise healthy people (p 214) If for some other reason quinine is injected it is advisable to remember these possibilities The initial injection should not exceed 0 2 gm (3 grs), this amount may be increased the next time to 0 5 gm (7½ grs) at most These larger amounts are administered only when the small dose was well tolerated Quinine or quinidine must be injected slowly

In recent years well tolerated quinine preparations for intramuscular injection have made intravenous injections superfluous One can inject 0 5 gm at a time intramuscularly without danger and if necessary this amount may be repeated once or twice on the same day The immediate action of an intravenous injection cannot be expected In most cases success is attained without recourse to injections , quinidine sulphate in amounts of 0 25 gm may be administered orally, with repetitions every two hours until the end of the attack, which usually occurs very soon

However, if the patient does not tolerate quinidine (by no means

a rare event in the presence of hypersensitivity) then digitalis and strophanthin are better alternatives The employment of these drugs in some cases affords such great advantages that they may be used instead of quinidine even when it is not contraindicated

If a rapid action is urgently necessary it is advisable to administer an intravenous injection of ¼ mg (1/250 gr) of ouabain The result is obtained in many cases in half an hour at the latest If the patient has not been previously treated with digitalis an injurious action need not be feared Even when the injection does not abolish the attack it may impair conduction and lead to periodic dropped beats or to a 2 1 block that is to a useful and considerable slowing of the ventricular rate

Previously ineffective carotid pressure may end a paroxysm if reapplied shortly after an injection of strophanthin This effect is comprehensible since the investigations of Hering and F Kisch have shown that the carotid sinus is sensitized by digitalis

In less urgent cases the oral administration of digitalis is recommended In this instance to be sure larger doses than are ordinarily employed in digitalis therapy are necessary as a rule It is advantageous to administer rapidly acting preparations (pure glycosides) in amounts which correspond to 0 4 to 0 6 gm (6 to 9 grs) a day of the assayed pulverized digitalis leaves If in the course of therapy of an auricular tachycardia a partial block between the auricle and ventricle develops often a marked reduction of the dose of digitalis succeeds in maintaining it Very often paroxysmal tachycardia vanishes completely during digitalis therapy In cases of *extra sys olie a paroxysmes tachycardiques* it is especially effective and as a rule superior to quinidine therapy

When a patient suffers from the paroxysms at regular intervals quinidine is preferable to digitalis because in this instance the attacks may be prevented by prophylactic treatment One proceeds exactly as in extrasystoles and endeavours to find the smallest amount sufficient to prevent the appearance of tachycardia

The combination of digitalis with physostigmine has often been recommended in order to reinforce that component of digitalis action which affects the vagus In this case physostigmine is administered orally in doses of 0 5 to 1 5 mg (1/120 to 1/40 gr) daily this dose should not be exceeded because of unpleasant untoward effects Prostigmine has also been recommended We are unable to perceive any definite advantages by the use of these drugs

Apart from quinidine, digitalis and strophanthin a large number

of other remedies have been recommended for the treatment of paroxysmal tachycardia It must be conceded that many other substances which act upon the myocardial cells can also arrest tachycardia This is true of atropine, but in large doses it produces unpleasant effects Acetyl β methylcholine may also be tried, it is effective also after hypodermic injection, but unfortunately it occasionally has unpleasant untoward effects Even apomorphine has been recommended to stimulate the vomiting centre and in this way the vagus But those who have once observed the unpleasant mode of action of apomorphine will no longer utilize this agent Ipecac in emetic doses is more worthy of recommendation (Weiss and Sprague) Vomiting is easily induced by "sticking the finger into the throat", it is a simpler procedure and just as effective The recommendation to treat tachycardias by adrenaline is incomprehensible, perhaps it occasionally abolishes the tachycardia but more often it may precipitate fatal ventricular fibrillation

Atabrine has also been recommended (Gertler and Yohalem) Vega Diaz could stop four out of six attacks of paroxysmal tachycardia or paroxysmal fibrillation by the intravenous injection of 0 1 gm of atabrine

Magnesium sulphate is effective in auricular tachycardia and in this condition it is not dangerous (Zwillinger) Ten to twenty cubic centimetres of a 15 per cent solution are slowly injected intravenously This agent is still in the trial period and additional extensive experience must be awaited Magnesium sulphate has been recommended particularly for tachycardias and extrasystoles following excessive digitalization As a matter of fact it does not act in every case and its effect is usually transient It has proved beneficial to us in paroxysmal auricular tachycardia Ventricular tachycardias will also respond favourably to the remedy We have obtained satisfactory results with an intravenous injection of 20 c c of a 20 per cent solution of magnesium sulphate

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XXII

DISTURBANCES OF STIMULUS CONDUCTION

INTRODUCTION, ESCAPED BEATS

THE capacity for conducting stimuli is a fundamental property which *every* myocardial fibre possesses regardless of whether it belongs to the common muscle or the specific tissue Every stimulus originating in, or applied to the cardiac muscle spreads over the heart, it is conducted as soon as it is above threshold

According to the prevailing conception conduction in the heart is myogenic In the excitation of a muscle fibre, electric depolarization appears on the limiting membranes The process of depolarization on one fibre acts as a stimulus to a neighbouring fibre etc In other words the excitation is transferred from one fibre to another Since a contraction is necessarily associated with the excitation of a healthy muscle fibre a contraction wave passes over the heart with the wave of excitation Actually with an appropriate experimental arrangement it has been possible to demonstrate in the ox's heart that after the auricular and before the ventricular contraction a contraction of the bundle of His and its branches takes place (Ishihara and Nomura)

In normal hearts the original stimulus is formed in the sinus node from which it spreads over both auricles through groups of muscle fibres that radiate in all directions By means of the A–V system the stimulus reaches the ventricles Since every area of the myocardium is capable of conduction a disturbance of this event may occur anywhere The results of this disturbance of conduction vary according to the site and size of the pathologic focus Naturally such a focus in the wall of the auricles will have much less significance than an equally large focus in the A–V system

As a matter of fact, disturbances of conduction are remarkably rare when one recalls how commonly myocardial diseases occur and how frequently one finds disturbances of stimulus formation (fibrillation extrasystoles) There are several reasons for this rarity A knowledge of the anatomy of the specific tissue assists in making this comprehensible

It was stressed earlier (p 11) that the sinus node is connected with the auricle by so many fibres that even when many of them become affected a sufficient number remain intact or functionally

efficient to keep the normal sequence of the heart beat from being disturbed For this reason sino auricular disturbances of conduction are rare

As stated earlier (p 16), the A–V system for the greater part of its course is separated from the common muscle moreover it possesses a special blood supply which comes from the right and left coronary arteries For this reason it has its own pathology Very often the ventricle is found thickly studded with scars or the cardiac muscle is severely degenerated (severe anæmia) while the A–V system is entirely normal On the other hand severe alterations are at times found exclusively in the specific system in otherwise normal hearts if for example only the arteries of the specific tissues are affected

Moreover anatomic investigations show that the right and left main trunks of the A–V system are built in an entirely different manner so that a small pathologic focus which renders the right bundle branch completely incapable of conduction only partly injures the left Furthermore the blood supply of the two bundle branches is entirely different The fact that the conduction system possesses excellent vascularization and that branches from the right and left coronary arteries anastomose with each other at many places is undoubtedly the reason why disturbances of conduction appear so seldom in spite of the great prevalence of myocardial diseases (which are mostly caused by a vascular disorder)

Not only these anatomic factors but also the physiologic properties of the heart muscle are responsible for the rare appearance of disturbances of conduction and (if such disturbances occur) for the lack of harmful results as a matter of fact the presence of disturbances often is not even noted

For a long time the opinion was advanced that conduction in the heart is undisturbed and proceeds with normal velocity only when a sufficiently wide path is available Narrowing of the diameter of the pathway in an area in the A–V system was supposed to lead to disturbances of conduction (Gaskell) Recent investigations performed with more modern methods however have shown that the width of the path does not determine the velocity of conduction and that as long as *one* fibre is intact in a cross section the conduction remains entirely normal

Only when this last fibre is also affected does a disturbance of conduction appear Conduction in the myocardium is called auxomeric this means that the stimulus can be transmitted from *one* fibre to any optional number (v Kries)

XXII

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healthy person (Lead III) The white perpendicular line indicates the beginning of carotid pressure The activity of the sinus node is immediately inhibited and deeper automatic centres assume control



Fig 203 Respiratory arrhythmia with escaped beats during bradycardia

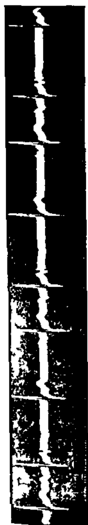


Fig 204 After two normal beats a blocked auricular extrasystole appears the long post extrasystolic pause is ended by an escaped beat

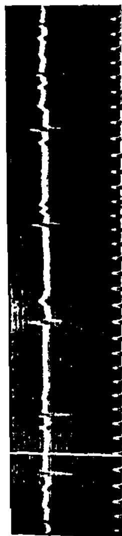


Fig 205 Sinus node activity is depressed by carotid pressure and escaped beats appear

in place of sinus stimuli thus preventing a prolonged ventricular standstill

Some very important forms of disturbance of conduction have

As a result, most profound lesions of the bundle of His need not produce disturbances of conduction as long as a single fibre in the cross section is intact

At times complete heart block appears overnight Nevertheless, histologic examination undertaken shortly after demonstrates the presence of very old and advanced lesions Conduction was normal as long as one fibre of the cross section remained intact, as soon as this became involved conduction was interrupted

But even when a complete interruption of the bundle of His prevents the stimulus from reaching the ventricle, the latter does *not stand still*, because the automatism of the deeper centres immediately becomes active Every specific fibre can form rhythmic *stimuli*, as discussed earlier (p 11), stimuli normally are formed *only in the head of the sinus node* and all the other specific fibres remain inactive If for some reason an interruption of conduction occurs, a centre situated below the site of the block immediately becomes active and assumes control as the most rapidly acting area of stimulus formation This automatism, acting as a protective mechanism, prevents standstill and thus prevents the patient from perceiving any unpleasant sensations despite heart block Only rarely does automatism fail (p 387)

In Fig 203 a marked respiratory arrhythmia may be seen The tracing was recorded in a child During expiration the activity of the sinus node was so slow that a stimulus from the A–V node became effective (p 398) During the slow rhythm beats occur which are not preceded by P waves These contractions, which serve to rescue the patient when stimuli from the higher centres fail to appear are called *escaped beats* These escaped beats are not pathologic but rather show that the automatism of the deeper centres is well developed

In Fig 204, after every two normal beats, an auricular extrasystole appears (it is recognized by the negative P wave coinciding with the T wave), it is not conducted to the ventricle but is blocked In the following post extrasystolic pause an escaped beat emerges from the A–V node, to be sure, some hundredths of a second later the post extrasystolic normal beat would have arrived, its P wave may be seen in the tracing between the QRS complex and the T wave of the escaped beat

Also during pressure on the carotid sinus, through which sinus node activity is depressed, the deeper centres lying in the A–V node may become active and prevent cardiac standstill

Fig 205 shows the effect of carotid pressure on the heart of a

conduction are presented by means of two drawings In Fig 206 the A-V system is indicated the other drawing (Fig 207) represents a chain of fibres of the A-V system Let us assume that the pathologic focus lies between the two horizontal lines of Fig 206 and the perpendiculars of Fig 207 Above and below to the right and left of these limits the conduction system is normal

Since a disturbance of conduction appears only if the disease impairs every fibre of the cross section a drawing of the type of Fig 207 is a permissible way indeed *according to the law of auxomeric conduction it is the only possible way* to explain abnormal conduction events

As soon as the affection begins in the tissues demarcated by the lines the last healthy muscle fibre 1 in Fig 207 will excite the first

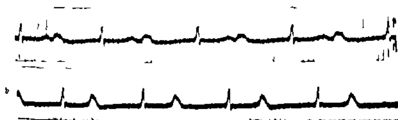


Fig 208 (Lead II) Two examples of a marked prolongation of conduction time In tracing b the P wave is concealed in the preceding wave

altered fibre 2 in a normal manner the pathologic fibre however responds to the stimulus slowly and likewise the stimulus response in general will be prolonged in the remaining diseased tissue But as soon as the stimulus again reaches healthy tissue (fibre 7) conduction will again proceed as rapidly as normal Owing to the retardation in the diseased tissue the ventricle will be excited later than normal and the auriculo ventricular conduction time the P–Q or P–R interval is prolonged *This prolongation of conduction time* is the first and simplest grade of disturbed conduction This retardation (prolongation of conduction delayed conduction) may be only trifling so that conduction may require 0 22 or 0 24 second but conduction times of 0 4 to 0 6 second and more are known But since *every* auricular stimulus reaches the ventricle even if slowly there is no disturbance of rate or rhythm

Examples of prolongation of conduction time are seen in Figs 188 and 201

In Fig 208a the conduction time is prolonged to 0 55 second so

already been considered In the initial chapters, the disturbances of intraventricular conduction were described, that is, disturbances of conduction below the site of division of the bundle of His (bundle branch block, arborization block, wide initial deflections and abnormal T waves) As a rule these are not characterized by a disturbance of auriculo ventricular beat sequence since even a complete interruption of conduction in the specific tissue of one ventricle leaves the connection to the other open Only in disease of *both* trunks is the path from the auricle blocked However this is rare

THE VARIOUS DISTURBANCES OF AURICULO-VENTRICULAR CONDUCTION

When a disturbance of conduction is mentioned one generally understands, in clinical parlance, a disturbance in the bundle of His above the bifurcation or in the A–V node The various forms of this

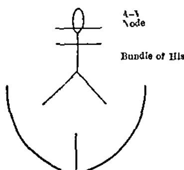


Fig 206 Schematic drawing of conduction system

disturbance of atrioventricular conduction will be considered in the following discussion

Let us assume that a pathologic focus develops in the A–V system for example in the course of rheumatic myocarditis With

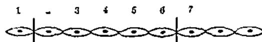


Fig 207 (Explanation in text)

increasing damage to the conduction path the passage of stimuli becomes increasingly difficult and the mildest form of disturbed conduction gradually is transformed into more severe disturbances

There exists an incomplete (partial) block as long as some stimuli still reach the ventricle from the auricle the block is complete when auriculo ventricular conduction is entirely interrupted

To facilitate comprehension the events in auriculo ventricular

periods are short and at times every third beat is dropped The auricles contract regularly

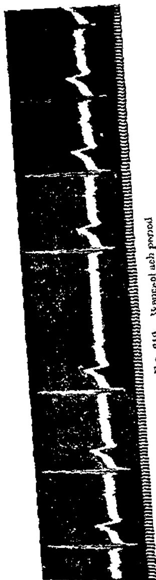


Fig 210 Wenckebach period



Fig 211 Paroxysmal auricular tachycardia with 2 1 block and Wenckebach period One stimulus is abnormally conducted within ventricle

In Fig 210 a longer period may be seen where every fifth to ninth beat is blocked The conduction time increases slowly The P waves are notched (disturbance of intra auricular conduction,

that a P wave appears soon after the T wave If the conduction time is longer or the rate faster, as in Fig 201 (P–R = 0 54 second), the P wave is concealed in the T wave (rarely even in the initial deflection of the preceding beat)

If the pathologic process progresses in the A–V system and the specific fibres themselves are more severely injured, it may happen that the first affected fibre (fibre 2 in the drawing) on occasion does not respond to the stimulus, and for this reason a stimulus is blocked on its way to the ventricle, accordingly a ventricular beat is dropped and the P wave is "blocked" Owing to the fact that the specific tissue did not conduct on one occasion, it may recover to such an extent that the stimulus following the blocked P wave is conducted much better—often normally, in fact But owing to fatigue of the

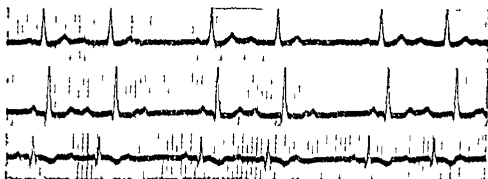


Fig 209 Wenckebach period

junctional fibres conduction time soon increases and after some time a block occurs This form of periodic dropped beat is called a Wenckebach period, since Wenckebach (1899) discovered it in an ingenious way by means of an analysis of the radial pulse alone A Wenckebach period is characterized by the following features despite rhythmic auricular activity periodically a ventricular beat is dropped the conduction time before the dropped beat being prolonged and after the dropped beat being shortened (owing to recovery) Such a period may be long and many beats may reach the ventricle before one is blocked but it may also be so short that every sixth fifth, or third beat may be blocked The length of a period may also vary continually in the same case The increase of conduction time may develop gradually or suddenly the conduction time may be greatly prolonged or may show only slight prolongation All possible variations occur

In Fig 209 a Wenckebach period is reproduced it appeared in a case of rheumatic myocarditis without digitalis therapy The

lock the stimulus may be conducted with normal or delayed elocity With further impairment of conduction nly every third or fourth stimulus is conducted 3 1 4 1 block etc)

Fig 212 shows a 2 1 block. Moreover there s a marked intraventricular disturbance of con duction with widening and slurring of the initial deflections

A 2 1 block also exists in Fig 213a At first glance it looks like a normal electrocardiogram It is suspicious however in that the form of the T waves resembles that of the P waves and that the P–T intervals are the same as the T–P intervals Since this may happen normally it cannot be employed as proof of a 2 1 block. In order to establish the diagnosis another tracing must be recorded after exercise or after inhalation of amyl nitrite

Fig 213b shows that auricular activity increased a little after the inhalation of amyl nitrite thereby the demands upon the conduc tion system were increased which thus became more easily fatigued so that a 3 1 block appeared If normal sinus rhythm had been present only a simple acceleration of rate would have occurred after amyl nitrite

A 2 1 block is present in Fig 214 On first inspection one gains the impression of blocked auricular extrasystoles The P–P interval which includes a ventricular beat is approximately 0 72 second long while those which appear subse quently are about 0 68 second The blocked P wave comes soon after the T wave and is premature But the same disturbances of auricular rhythm are occasionally found in 2 1 block or with complete block since the interval between two auricular waves which includes a ventricular systole is often shorter than one which does not No satisfactory explanation for this phenomenon has yet been put forward (Kauf) A vagal reflex seems to exist since the systolic increase of blood pressure brought about by the aortic and carotid nerves leads

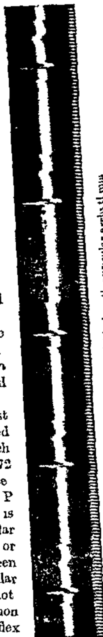


Fig 214 (Lead II) 2 1 block with auricular arrhythmia

p 381) The tracing was obtained from a patient with a mitral valve lesion who had been treated with massive doses of digitalis

In Fig 211 there is a paroxysmal auricular tachycardia with a disturbance of conduction from auricle to ventricle In tachycardias, just as in auricular flutter, these disturbances of conduction



Fig 212 (Lead II) 2 1 block and disturbance of intraventricular conduction

are common The auricular rate amounts to 187 At the beginning of the tracing there is a 2 1 block, then one beat is conducted to the ventricle quickly, the next one slowly and aberrantly (a branch of the conduction system below the bifurcation of the bundle was not functioning), only the third stimulus is blocked Thereafter a 2 1 block occurs and then another short period This disturbance

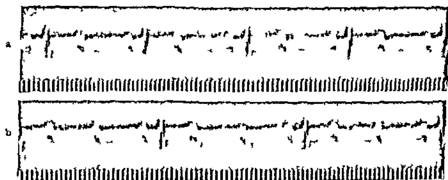


Fig 213 (Lead II) There is a 2 1 block in (a) which is transformed into a 3 1 block in (b) after inhalation of amyl nitrite

of conduction immediately vanished when the tachycardia reverted to sinus rhythm

In the shortest possible period every third beat is dropped The conduction of the second stimulus is prolonged in comparison to the first and the third beat is blocked If the disturbance increases then the bundle becomes fatigued even after the passage of one stimulus so that the next one is not conducted a 2 1 block ensues and every second stimulus is not conducted In a 2 1

block the stimulus may be conducted with normal or delayed velocity With further impairment of conduction only every third or fourth stimulus is conducted (3 1 4 1 block etc)

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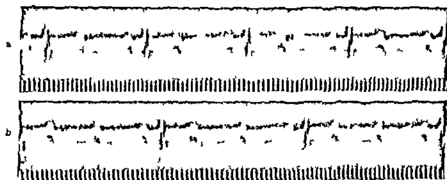


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Fig 16 Block than 2 1 block In long ventricular pauses a deep ventricular center is active



Fig 17 Complete auriculo ventricular block



Fig 218 Complete auriculo ventricular block

to an increased vagal tone which becomes manifest only during the following cycle Fig 215 shows a regular 4 1 block (Lead III)

Much higher grades of block are also possible, for example, there may be 10 1 block in which only one of ten stimuli is conducted from the auricle to the ventricle, after which the bundle recovers so slowly that the following nine beats are blocked In high grade block the ventricles naturally do not remain inactive until the fifth or tenth stimulus is conducted In marked heart block (rarely in 3 1, but usually in 4 1) the deeper centres become active during the longer ventricular cycles A centre lying below the area of block develops its own automatism, and since it is no longer depressed by more frequently conducted stimuli it may assume control of the ventricle

In Fig 216 a 7 1 block is seen followed by a 2 1 block Every seventh and later, every second auricular stimulus reaches the ventricle with a conduction time prolonged to 0 30 second The



Fig 215 4 1 block

depressed RS–T interval and the inverted T wave of the conducted beats (Lead II) indicate myocardial damage In the long pauses created by the block a more deeply situated automatic centre begins to function Since the stimuli formed by this abnormally situated centre spread abnormally within the ventricle the related ventricular complexes appear altered

If the conduction to the ventricle fails entirely the auricles beat, as under normal conditions in sinus rhythm but the ventricles contract with complete independence under the control of their own automatic centre In this instance there is a complete block or a *total dissociation of the two rhythms*

In an electrocardiogram complete block is recognized by the regular auricular and regular ventricular activity but *continual variation of the P–R interval* As will be shown later the rate of the ventricle may vary

In Fig 217 the auricles and ventricles contract with perfect regularity The auriculo ventricular (P–R) intervals vary continually so that it must be assumed that the ventricles work independently of the auricles and with their own rhythm In other

in extrasystoles tachycardias flutter, and fibrillation of the auricles are described in their respective sections (pp 264 and 294)

In other words it is evident that a definite period of recovery is necessary for normal conduction The more profoundly conduction is impaired the longer this recovery time must be For this reason it is comprehensible that disturbances of conduction appear much later in bradycardias than in tachycardias In a combination of an auricular tachycardia and injury to the A–V system disturbances of conduction appear very regularly and early Disturbances of conduction may vanish when the heart rate slows

Besides these disturbances of conduction in which the pathway is normal there exist two other groups in which the conduction path is actually damaged

Disturbances Due to Drugs There are disturbances of conduction from the effect of drugs on the A–V system As an example digitalis deserves particular emphasis Other agents scarcely come under consideration in the clinic For a long time it was believed that salicylates could produce disturbances of conduction but more recent investigations have shown that even when salicylates are given in large amounts they do not influence conduction in the heart

Digitalis impairs conduction in two ways It acts upon the muscle itself reducing the irritability of the fibres and prolonging their refractory phase Moreover it depresses conduction through an increase of vagal tone (p 303) All degrees of incomplete and complete block can develop from the effect of digitalis

The status of the heart muscle is of importance in regard to the appearance of digitalis extrasystoles this is equally true of disturbances of conduction While very large amounts of digitalis are necessary to induce disturbances of conduction during health in a cardiac patient at times they occur even after small doses There are patients who show a disturbance of conduction even after three tablets of 0 1 gm of digitalis

If digitalis therapy is continued in spite of the appearance of a disturbance of conduction there are two possibilities Either it increases and a higher degree of block appears or the disturbance vanishes and gives way to normal rhythm Since the status of the heart muscle plays an essential role in the occurrence of digitalis conduction disturbances it is possible that the improvement accompanying a progressive digitalization removes the sensitivity of the conduction tissue to digitalis and thus the existing conduction disturbance may vanish In other words a behaviour similar to

Clinical Aspects of Disturbances of Auriculoventricular Conduction

The etiology of auriculoventricular conduction disturbances may vary considerably It is convenient to differentiate between three groups —

1 So called functional disturbances of conduction
2 Disturbances of conduction due to poisons
3 Disturbances of conduction due to organic disease

Functional Disturbances There are disturbances of conduction in a perfectly healthy heart These are called—not very aptly—*functional disturbances of conduction* They are found in conjunction with various disturbances of rhythm, and are essentially dependent upon two factors

They appear when demands are made upon the conduction path very early in diastole this occurs not rarely for example in extrasystoles Reference has previously been made to the fact that auricular extrasystoles may be blocked or may be conducted aberrantly if they are very premature These disturbances of conduction are not an indication of disease they occur in perfectly healthy hearts, since the heart and conduction tissues although healthy have a refractory period and for this reason require a certain amount of time for recovery If the auriculoventricular conduction system is stimulated prematurely it conducts either poorly or not at all

Conduction disturbances also appear when too many stimuli per minute are presented for conduction The functional capacity of a conduction pathway permits response to demands only up to a *certain limit It can be shown experimentally that when a demand* is made upon the bundle of His to conduct only 100 beats per minute, the conduction time increases a little (Lewis and Master) But if higher rates occur, as in a tachycardia or if auricular flutter develops, with an auricular rate of 300 per minute or auricular fibrillation, with a rate of 600 the conduction path between the auricle and ventricle is fatigued by overwork Naturally it is very advantageous for the patient when the ventricle does not respond with a high rate If in auricular fibrillation all the auricular stimuli reached the ventricle *death would follow immediately In auricular* fibrillation an incomplete auriculoventricular block always exists as soon as the fibrillation or tachycardia ceases auriculoventricular conduction again becomes normal These functional disturbances

may be blamed In older individuals coronary sclerosis is most *often responsible* It should be stressed once again that a disease of the common muscle need not always involve the specific tissue and *conversely* that the specific tissue may be severely affected *although* the working muscle is normal These events are explained by the isolation of the conduction tissue from the common muscle and by the fact that the specific tissue has its special blood supply

When the history reveals the absence of previous digitalis therapy a careful investigation must be undertaken to determine which *organic* cause is responsible for the disturbance of conduction

The clinical diagnosis of many forms of disturbances of conduction is not possible without the employment of graphic methods In a simple prolongation of auriculoventricular conduction there is no alteration of the rate or rhythm to make the diagnosis possible But not rarely attention is drawn to the long conduction time through the appearance of gallop rhythm The auricles also produce sounds which are normally inaudible since they merge with the first heart sound In prolongation of conduction time the *auricular sound occurs* so early in *diastole that it becomes audible* as a third heart sound before the two normal sounds This gallop rhythm vanishes immediately when conduction again becomes normal

Wenckebach s periods can at least be suspected by clinical examination under the following conditions when a patient with rhythmic cardiac action has received digitalis or has recently had diphtheria or a myocardial disease and suddenly manifests periodic omissions of ventricular systoles in contrast with extrasystoles the pauses caused by dropped beats are not preceded by premature contractions

In most cases the diagnosis of a complete heart block also is possible without an electrocardiogram In the first place it is important to know that in heart block a bradycardia need not always exist In former years heart block was considered only when a decided *bradycardia* was present and it was assumed that every case *of heart block manifested a slow heart* rate But it has been pointed out that in complete block a fast ventricular rate may be present and for reasons mentioned later this is very commonly the case In other words bradycardia is not the *chief sign of* complete block

Even when a marked bradycardia exists heart block need not

that already discussed in digitalis extrasystoles is also encountered in this connection

Disturbances of conduction which appear during digitalis therapy do not contraindicate its continuation An incomplete block may be increased or transformed into a complete block But this does not constitute a danger, since even with high grade block and with complete interruption of conduction between the auricles and ventricles a normal circulation may exist

Moreover, it is incorrect to withhold digitalis from a patient needing it simply because a disturbance of conduction from other causes is present If for any reason digitalis is indicated it should be given despite disturbances of conduction Indeed in patients with auricular fibrillation an endeavour is regularly being made to produce an increase of the degree of block by means of digitalis *those* cases of auricular fibrillation which exhibit a severe disturbance of conduction after small doses of digitalis for this reason often respond particularly well to digitalis therapy It would be incomprehensible if the production of conduction disturbances were aimed at in auricular fibrillation but were feared as a complication in sinus rhythm

Digitalis can produce all forms of incomplete and even complete heart block For this reason in every patient with a disturbance of conduction an inquiry should be made concerning previous digitalis therapy The disturbance may persist for several days after the cessation of treatment

Disturbances Due to Organic Disease One last form of disturbance of conduction depends upon an organic disease of the A–V system It is not a very rare event to find a congenital heart block in patients with a congenital defect of the ventricular septum The bundle of His lies in the immediate neighbourhood of the septum membranaceum In the absence of parts of the septum and its adjacent muscles the anlage of the bundle of His may be disturbed

A structural injury of the conduction system may appear in the course of a toxic or degenerative diffuse damage of the heart muscle , it may be caused by foci located in the conduction system or, finally, it may be the result of a disease of the vessels which nourish the specific tissues All kinds of pathologic processes in the myocardium may also appear in the specific tissues , among those which may be present are diphtheria rheumatic myocarditis gumma tuberculosis, myomalacia and carcinomatous metastasis In children and young people diphtheria or rheumatic disease usually

may be blamed in older individuals coronary sclerosis is most often responsible It should be stressed once again that a disease of the common muscle need not always involve the specific tissue and conversely that the specific tissue may be severely affected although the working muscle is normal These events are explained by the isolation of the conduction tissue from the common muscle and by the fact that the specific tissue has its special blood supply

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Even when a marked bradycardia exists heart block need not

Fig 221a shows complete heart block with a ventricular rate of 84 The block developed during digitalis therapy After five drops of amyl nitrite the complete block vanished, and although a prolonged conduction time remained (221b), all stimuli reached the ventricle A few minutes later the heart block reappeared and vanished finally only after the discontinuance of digitalis therapy The tracing was obtained from a case of severe coronary sclerosis The automatic beats during the heart block (221a) are identical with the beats conducted after amyl nitrite this favours the assumption that the automatic beats originated above the site of division of the bundle

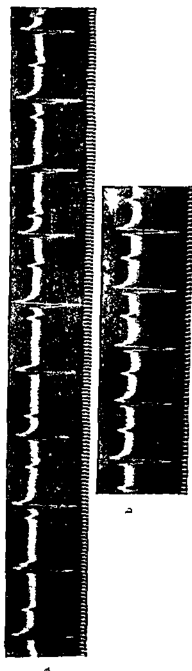


Fig 221 In (a) a complete auriculoventricular block exists which after inhalation of amyl nitrite passes over (b) into full rhythm with prolonged conduction time

Cases of partial heart block react very differently to physical exertion The block may become increased or diminished The sympathetic tone increases after exercise and thus improves conduction But simultaneously the auricular rate is increased thus the conduction pathway is subjected to greater demands and auriculoventricular conduction is impaired According to the factor which predominates the existing block will be increased or diminished by work An example of the increase of block by effort is furnished by Fig 213

The assumption of Mobitz that Wenckebach's periods occur only in the A–V node was refuted by the experiments of Scherf and Shookhoff who succeeded in animal experiments on the heart *in situ* in injuring the bundle branch at an area remote from the A–V node and in producing periodicity

Subsequently Schellong produced Wenckebach's periods in muscle strips by digitalis intoxication

There is one form of partial heart block (i e of periodic dropped beats) which is never the result of *increased* vagal tone of a general intoxication or of digitalization but is always due to a circumscribed anatomic cardiac disease It is differentiated from the Wenckebach period by the fact that the conduction time remains constant that is *no* increase of conduction time occurs in a long series of conducted impulses for no progressive change takes place Moreover at times several auricular beats may be blocked in succession this never occurs *in a Wenckebach period* where only one stimulus is blocked It is certain that the intensity and extent of the pathologic process *in* the specific tissue and not the site of the pathologic process decides which form of periodic dropped beats occurs

This form of periodic dropped beats which has just been described has been called by Mobitz Type II of periodic dropped beats in order to differentiate it from the Wenckebach period or Type I

Clinical and experimental observations support the suggestion that Type I periodic dropped beats appear in extensive injury of the conduction system while those of Type II are found only in circumscribed injuries In intoxications for example with digitalis or fatigue of the conduction pathways (tachycardias) only Type I (Wenckebach's period) is found

In Fig 222 at the beginning of Lead I only two of three auricular stimuli are conducted to the ventricle (3 2 block) In contrast to a Wenckebach period with 3 2 block (see Fig 209) the conduction time remains constant Then a group of four auricular stimuli follows of these only two are conducted to the ventricle (4 2 block) Despite the great differences in the duration of the ventricular pauses here also the P-R intervals remain unaltered The same is evident in other leads

Apart from the auriculoventricular block of Type II a severe disturbance of intraventricular conduction is present in Fig 222 The ventricular complexes show the picture of left bundle branch block

The complaints of patients with disturbances of conduction are remarkably few In most cases the patient does not become aware of the abnormal state In prolongation of auriculoventricular conduction the cardiac rate and rhythm on the whole are not disturbed In cases of Wenckebach periods the pause conditioned

by the failure of conduction is not sufficiently long to evoke symptoms The pause is hardly ever perceived In higher grades of block the deeper centres become active during the longer ventricular pauses, and through their own rhythm prevent any unpleasant stand still Reference has been made elsewhere to the fact that in complete failure of auriculoventricular conduction the centres of the ventricle assume control, and the patient generally does not notice this severe disturbance *Only when automatism fails for reasons considered below does the patient become conscious of the disturbance*

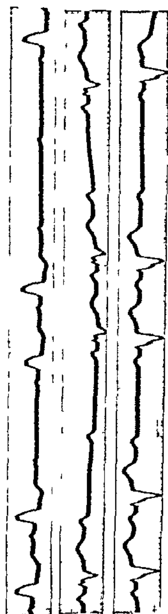


Fig 222 Type II of periodic dropped beats and disturbance of intraventricular conduction

The rate of automatism of the ventricles in many cases of heart block is as has been mentioned very high Minute rates exceeding 60 are very common Moreover rates of 80, 100 and more have been observed (Fig 221) The explanation of this becomes evident by the following consideration the A–V node represents a physiologic barrier for auriculoventricular conduction even *normally it retards auricular stimuli* Thus it is conceivable that in heart block due to an increase of vagal tone intoxications, or one of many organic disturbances of conduction the site of block is high in the A–V node If the interruption of conduction occurs quite high in the node an automatic centre highly situated and just below the site of block becomes active This A–V nodal centre has a *highly developed automatism* As a rule its rate is only 15 or 20 beats per minute slower than that of the sinus node

If in heart block the ventricle beats slowly, two factors may be responsible 1 The interruption of conduction may be located so low that a very deep automatic centre becomes active In a deeply situated block the ventricular automatism can be as low as 30 beats

or less per minute When auriculoventricular conduction is interrupted by a bilateral bundle branch block the automatism may be even less in rare cases rates of 16 or even 10 beats per minute have been observed (see Fig 218) In complete auriculoventricular block caused by a bilateral bundle branch block variform ventricular extrasystoles frequently occur This phenomenon is understandable since the same lesion which causes the block may irritate the specific fibres and thus initiate abnormal stimulus formation

2 One can also observe a slow automatism in a highly placed block when the pathologic process which leads to block also injures the deeper automatic centres Under these circumstances they cannot become active with their customary rate and therefore work very slowly

It is interesting that in many instances patients who show a ventricular automatism of 30 or less also have strikingly few complaints as long as they do not exert themselves unduly A wonderful compensating mechanism in the circulatory dynamics prevents disturbances It was emphasized earlier that a bradycardia is very commonly found in trained athletes The slower the cardiac rate is the larger the stroke volume If the stroke volume with a minute rate of 80 amounts to 60 c c with 40 beats per minute it may be 120 c c For the circulation only the product of these two factors the minute volume has significance it is obvious that this may be just as great with a bradycardia of 40 as with a rate of 80 since diastole is longer and the ventricle fills better when the cardiac rate is slow (Lundsgaard) In other words in bradycardias providing they do not exceed a certain limit all disturbances are completely compensated and clinical experience shows that this holds for rates down to 20 beats per minute as long as the heart and the vessels are otherwise healthy

In very decided bradycardias the heart is enlarged by virtue of the greater filling One often reads in X ray reports of athletes and cases of heart block Plump universally enlarged heart However the dilatation is dynamic in origin On auscultation of these patients as a rule a systolic murmur is audible over the aorta probably because the dilatation of the left ventricle makes the normal aortic orifice act as a *relative stenosis and* because of the increased output Frequently the systolic blood pressure is increased in these cases of heart block and bradycardia particularly in elderly patients

With greater exertion to be sure the circulation of these cases of heart block all too easily becomes inadequate In the healthy

person with the heart beating in sinus rhythm, the cardiac rate is increased by the performance of work through alteration of the vagal and sympathetic tone, so that the minute volume increases somewhat and the musculature receives more blood The situation is different in a deep seated heart block The vagus on the whole has no influence on the centres located deep in the ventricles (p 24) and the accelerating action of the sympathetics is likewise much less than on the higher centres, accordingly, after work, the cardiac rate remains far below the actual need Vertigo, dyspnœa and states of anxiety then appear

The patient must adapt his performance to the condition of his circulatory system If he does this he may remain free from symptoms for years Patients with heart block who engage in heavy physical work without any complaints are commonly encountered Unpleasant events occur only when Stokes Adams attacks appear (p 387)

The appearance of a disturbance of conduction however, has great significance because in patients who have not undergone previous digitalization it permits one to assume a myocardial disease even though all other signs are absent The finding of a disturbance of conduction to be sure, at first shows merely that a pathologic focus exists in the A–V system Whether it involves an acute or chronic disease, an inflammation, a necrosis, an old scar, or degeneration can be determined only by the clinical examination *In an old person a simple prolongation of conduction time may be* the first indication of the presence of coronary sclerosis and its appearance in rheumatic disease may indicate the presence of myocarditis Like abnormal T waves in many cases the development of conduction disturbances alone permits the diagnosis of a myocardial disease

Such alterations are often transient and may be absent during a single examination If an electrocardiogram is recorded on alternate days on the average every second case of acute rheumatic fever will exhibit disturbances of conduction The disturbance can also be the sequel to a recent diphtheria or rheumatic myocardial disease that has been inactive for years It may lack significance if the common muscle is intact Despite the presence of heart block these patients can pursue an active life without symptoms or treatment The freedom *from symptoms of most* patients having conduction disturbances is an important reason why this condition is diagnosed so rarely indeed it is usually an accidental finding

The prognosis is provided by the underlying pathology and not by the discovery of a disturbance of conduction alone

The treatment of a disturbance of conduction may be omitted in most cases If it is asymptomatic one should not be alarmed by it and should direct attention only to the underlying pathologic process

Disturbances of conduction caused by digitalis subside rapidly when the drug is omitted If an organic disease of the conduction system exists only rarely can it be influenced by medication No remedy is known which prevents diphtheritic necrosis and none for restraining the progress of coronary sclerosis But an endeavour should be made to improve coronary circulation by the administration of vasodilating remedies (preparations of theophylline papaverine) Even when a gumma has produced heart block specific treatment rarely yields a result since the retrogression of a gumma also leaves a scar which usually impairs auriculoventricular conduction If the disturbance is due to an old scar all therapy will be useless from the start

In former years treatment was recommended for *every* case of block and atropine was often given for this purpose In this connection it may be said that the effective dose of atropine also produces untoward actions and its continuous administration is impossible

Thus it happens that only extreme bradycardias and attacks of Stokes Adams require therapy the remedies employed are mentioned later (p 392) one should not reach immediately for the prescription pad in every disturbance of conduction

INTRA AURICULAR DISTURBANCES OF CONDUCTION

Just as disturbances of intraventricular conduction are recognized by notching and widening of the QRS complexes intra auricular disturbances of conduction may be assumed when notching and widening of the P waves are found Owing to the presence of pathologic foci in the auricles or ramifications of the sinus node the wave of excitation is compelled to spread abnormally from its site of origin in the sinus node The abnormal P waves may be permanent or merely transient according to the type of conduction disturbance

Abnormal P waves naturally can also result from an abnormal origin of excitation (abnormal site of stimulus production in the auricle auricular extrasystoles) just as widened ventricular com

plexes occur not only in disturbances of intraventricular conduction but also in ventricular automatism or in ventricular extrasystoles The finding of variform P waves with normal auricular rhythm and constant P–R intervals is in favour of an intra auricular disturbance of conduction and against a disturbance of stimulus formation (Scherf and Shookhoff, Rothberger and Scherf)

Apart from slurring and notching P waves may also exhibit an alteration of form in the sense of increased amplitude (more than 2 5 mm in height) and flattening, they may become inverted and wider (more than 0 11 second) Just as the QRS complex develops from the sum of potentials of the two ventricles, the normal P waves represent a summation of potentials developing from the excitation of *both* auricles Since the sinus node is located in the right auricle, this chamber is stimulated earlier (0 01 second) than the left, so that a slight difference in time normally exists between the excitation and contraction of the two auricles From a negative P wave one may not assume an origin of excitation in the A–V node, although a stimulus originates in the head of the sinus node, at a normal site inverted P waves may result from abnormal spread of the excitation

Abnormally large and notched P waves are a frequent finding in mitral stenosis probably in connection with the dilatation of the left auricle (see Fig 271a)

According to Winternitz the P wave in cor pulmonale is heightened only in Leads II and III and is neither widened nor notched, it is low in Lead I In mitral lesions the largest P waves are found in Leads I and II They are usually notched and widened The authors can confirm this statement It is not as yet determined whether these changes are caused by hypertrophy alone or also by dilatation Some observations support the assumption that the abnormal P waves of cor pulmonale are caused by hypertrophy while dilatation with intra auricular block is responsible for them in mitral cases Ippolito and Reinstein confirmed these findings

Fig 223 shows three electrocardiograms obtained from patients with pulmonary disease The first tracing was registered on a patient with chronic fibroid pulmonary tuberculosis while the second and third tracings were obtained from two patients with pulmonary emphysema They show different degrees of right axis deviation and the T waves in the first and second case suggest the presence of right ventricular strain The P waves are typical for cases of this kind They are very small in Lead I and unusually high but not widened in Leads II and III

Isolated disturbances of intra auricular conduction are rare, more frequently they are found in conjunction with disturbances of atrioventricular or intraventricular conduction Under these circumstances they are significant because they indicate that a pathologic focus exists not only in the ventricle but al o in the auricle and that a widespread disease is present

Examples of disturbances of intra auricular conduction are seen in Figs 29 and 145

In very rare cases a complete di sociation is found between the two auricles (Scherf and Siedek) The disturbance can be produced

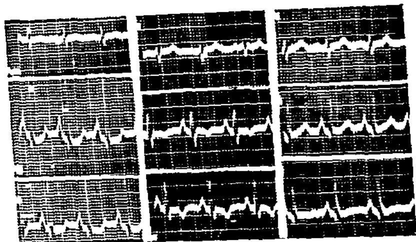


FIG 3 Three cases of cor pulmonale with evidence of righ t ventricular strain and typical alterations of P waves

experimentally In this instance one finds two auricular rhythms completely independent of each other (interauricular block)

According to Wood and Selzer widened P waves of low voltage and flat topped or bifid ones are indicative of left ventricular failure and imply left auricular stress

Large P waves particularly in Lead II are not rare in congenital heart lesions particularly in the tetralogy of Fallot

Since the left auricle becomes depolarized later than the right auricle delay in the activation of the right auricle will not lead to a widening of the P waves (Spuehler)

In some sino auricular conduction disturbances the P–R interval may be short This is said to happen when conduction from the auricle to the ventricle is normal but the conduction from the sinus node to the main parts of the auricle is delayed (Spuehler)

SINO-AURICULAR DISTURBANCES OF CONDUCTION

Sino auricular conduction disturbances are rare chiefly because of the presence of a great many connections between the *sinus node* and the auricle and owing to the large dimensions of the sinus *node*

Moreover every single fibre of the sinus node is capable of *forming stimuli*, so that with destruction of large parts sufficient tissue capable of function still remains Most cases of sinus block are produced by digitalis therapy, this is comprehensible, because digitalis acts upon all centres and all paths of conduction *There is experimental* evidence for the appearance of sino auricular block as a consequence of intracardiac reflexes elicited by focal stimuli in the auricle (Scherf)

In addition to sinus block caused by failure of conduction of the stimulus, there is a type in which the stimulus is not formed at all This type is rare More commonly sinus block can be traced to a disturbance of conduction with normal stimulus formation Since *it has not been possible* as yet to register the electrogram directly from the sinus node in cases with sino auricular block *a decision* between non formation or non conduction of a stimulus is often difficult

Disturbances of conduction between the sinus node and the auricle are fundamentally similar to those that exist between the *auricle* and *the* ventricle The most common and also the most easily recognized form is a periodic omission of systole (dropped beat) through a block of Type II One or more systoles (of the entire heart auricle *and* ventricle) are dropped since the corresponding number of stimuli is not conducted beyond the sinus node Thus it results in the appearance of complete cardiac pauses whose duration is double, triple, or some multiple of a cardiac cycle If these pauses are longer the deeper A–V centres become active if this does not occur because the centres are damaged Stokes Adams attacks appear In the presence of higher grades of sinus block and as the result of the repeated appearance of A–V beats at times a remarkable tracing develops

If a complete sinus block exists (that is if no normal stimuli pass from the auricle to the ventricle) the A–V node is continually active and A–V rhythm appears (p 398)

Fig 224 shows pauses which are exactly double the length of normal periods in an otherwise regular heart During these pauses not only the ventricle fails to contract but the auricle is also inactive

In Fig 225 four beats are dropped soon after the beginning, the pause amounts to five times the normal period (5 08 seconds) quite late (only after the lapse of 4 5 seconds) an automatic beat occurs

In Fig 226 a very marked but regular sinus bradycardia of 27 beats per minute (length of cycle 2 20 seconds) is present This bradycardia is due to the fact that every other sinus stimulus is not conducted (or not formed?) In the long pauses an A-V nodal centre becomes active so that a very peculiar tracing develops In the T wave of the first automatic beat a normal P wave is seen which is not followed by a ventricular complex since it occurred too early then an automatic beat again appears and soon thereafter a sinus beat with entirely normal conduction Owing to the



Fig 4 (Lead I) Sino auricular block

sinus bradycardia an automatic beat again follows in whose T wave is concealed a P wave which is slowly conducted to the ventricle The next P wave is concealed in the T wave of the following automatic beat and is blocked

In Fig 227 at first glance one might diagnose extrasystoles On closer inspection another disturbance is recognized

A sinus bradycardia of 42 beats per minute is present The first P wave in Fig 227 is seen after the first QRS complex it is visible in the T wave the next P wave follows immediately behind the third QRS complex and adjoins immediately on an S wave the third P wave follows the fourth QRS complex and has the same position in the cycle as the first P wave the beats of abnormal form which always follow 0 24 to 0 28 second after a P wave are ventricular complexes of conducted beats (not extrasystoles) Their abnormal form is the result of their prematurity (aberrant conduction in the ventricle) Owing to the marked sinus bradycardia which again is probably the result of a 2 1 sino auricular block escaped beats from the A-V node repeatedly occur

Thus the same disturbance is present as the one reproduced in Fig 226 The peculiar appearance of Fig 227 is merely the result of a different grouping occasioned by other rates of the sinus and



Fig. 225 (Lead II). Sino auricular block with an escaped beat at end of cardiac standstill

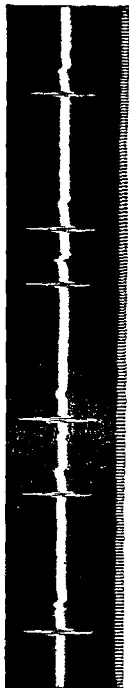


Fig. 226. Sinus bradycardia from sinus block (2:1) with intrusion of escaped beats



Fig. 227. Extreme sinus bradycardia through 2:1 sinus block with escaped beats. Conducted sinus beats are entirely aberrant owing to abnormal spread within ventricle

of the escape rhythm. Likewise the marked bradycardia in Fig. 38 is probably the result of a 2:1 or 3:1 sinus block. These tracings can be interpreted as due to interference dissociation (p. 402)

In sinus block the pauses caused by dropped beats do not always

constitute an exact multiple of a normal period since the sino auricular conduction before the dropped beats is prolonged and afterward may be shortened (as in A–V block) Indeed in a Wenckebach s period the pause *may be much shorter* than double the normal period Exact analysis of such sinus arrhythmias which are independent of *respiration* is impossible because the activity of the sinus node cannot be directly registered that is changes of the sino auricular *conduction* are not demonstrable For the same reasons a diagnosis of a simple prolongation of sino auricular conduction—*without dropped* beats—cannot be made since such electrocardiograms cannot be differentiated from normal ones

The treatment of sinus block is arranged in accordance with the same viewpoints as the treatment of disturbances of auriculo ventricular conduction *In a majority* of sinus blocks of organic origin an affection (sclerosis) of the arteries supplying the node exists

STOKES ADAMS SYNDROME

The syndrome named after Stokes and Adams[1] is observed when for some reason the circulation stands still and the supply of blood to the brain stops If this standstill lasts only three or four seconds usually it is not noticed by the patient only a pause which persists for a longer time causes a sensation of vertigo after approximately ten seconds unconsciousness occurs and the patient faints with longer standstill there is twitching of the arms and legs and finally tonic and clonic convulsions Urine and stool pass involuntarily A standstill persisting for longer than three or four minutes is fatal

This event can be evoked by two mechanisms which are fundamentally different from each other their distinction is important in respect to treatment

The first major type formerly regarded as unusual is much more common than the second and according to recent investigations is caused by tachycardia It was stated earlier (p 300) that the higher the rate and the shorter the diastole the smaller the stroke volume If the cardiac rate exceeds a certain limit the time available for ventricular filling becomes too short and the ventricle expels too little blood with too little force so that practically the circulation stands still With a powerful myocardium and with normal vessels otherwise healthy young individuals will tolerate a remarkably high

[1] It was also described by Morgagni



FIG 225 (Lead II) Sino auricular block with an escaped beat at end of cardiac standstill

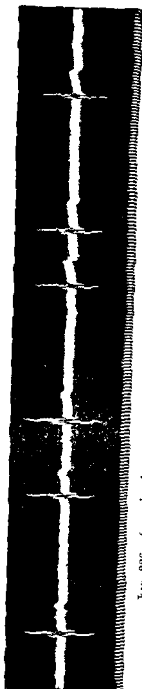


FIG 226 Sinus bradycardia from sinus block (2 1) with intrusion of escaped beats

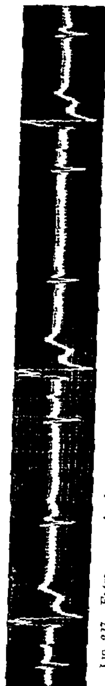


FIG 227 Extreme sinus bradycardia through 2 1 sinus block with escaped beats Conducted sinus beats are entirely aberrant owing to abnormal spread within ventricle

of the escape rhythm Likewise the marked bradycardia in Fig 38 is probably the result of a 2 1 or 3 1 sinus block These tracings can be interpreted as due to interference dissociation (p 402)

In sinus block the pauses caused by dropped beats do not always

(for example if arteriosclerosis produces not only the block but also damages the deeper centres) then a pause before the institution of automatism produces a Stokes Adams attack Since complete heart block after its first appearance usually does not persist uninterruptedly as the conduction temporarily recovers until it again fails new attacks occur repeatedly In the history of patients with heart block the report is frequently heard that months or years ago frequent attacks of vertigo and fainting occurred One should realize that heart block developed precisely at that time

Fig 228a shows the beginning and Fig 228b the end of a Stokes Adams attack due to ventricular standstill Since it is impossible to employ leads from the extremities during convulsions a chest lead was used The record was obtained from the right second and fifth intercostal space parasternally In this way remarkably high P waves are obtained which at first glance might be confused with ventricular complexes They are followed by a negative after deflection the Ta wave In the upper tracing the second complex is a QRS complex Two QRS complexes are visible in the bottom tracing

At the beginning of the tracing in Fig 228 a ventricular complex may be seen between two auricular waves then all ventricular action is absent for one minute and thirty six seconds The auricle beats even

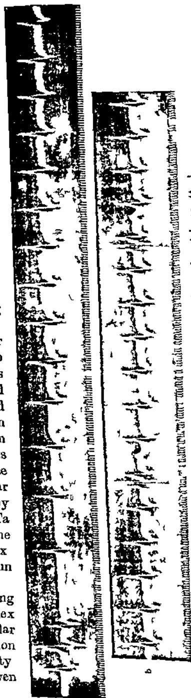


Fig 228 (chest lead) Beginning (a) and end (b) of a Stokes Adams attack

rate extremely well Thus we observed a young man with a tachycardia of 326 beats per minute who was still able to walk slowly to the clinic In older patients with sclerotic vessels in severe heart muscle disease and in valvular lesions with predominant stenosis of an ostium (and, for this reason, a small stroke volume from the start, even with a lower rate of the heart) the circulation may become inadequate for satisfactory perfusion of the central nervous system In a severely damaged heart due to coronary thrombosis even a tachycardia of 180 beats per minute may produce unconsciousness Very often when this happens we are dealing with paroxysmal auricular fibrillation in old people with sclerosis of the smaller vessels (p 302) Rarely ventricular fibrillation exists Paroxysmal ventricular fibrillation has been reported to occur in patients without apparent organic heart disease (Moe, Storstein)

Frequently, despite loss of consciousness, a slight circulation continues, since the heart still advances a small amount of blood, so that even a longer duration of the attack is not fatal

The second main form of Stokes Adams is encountered in actual cardiac standstill There are various subgroups Most commonly a ventricular standstill appears in disturbances of auriculoventricular conduction when the impulse is not transmitted to the ventricle and the deeper centres do not assume immediate control over the ventricle automatically Normally the deeper centres are prepared to step in immediately upon failure of conduction of stimuli to the ventricles If one sections the bundle of His in an exposed dog heart slight cardiac slowing is seen, but no pause occurs since an automatic centre below the division becomes immediately active and prevents standstill But if this experiment is performed on another animal with the difference that prior to the severing of the path of auriculoventricular conduction a small amount of quinine is injected and thus the automatism of the centres is impaired then they do not function immediately after the section rather a certain period elapses until sufficient stimulating metabolites have accumulated in the centres This pause before the beginning of automatism is called the pre automatic pause The more the specific fibres are damaged the longer the standstill The longer ventricular standstill lasts, the more severe its effects

Corresponding to these experimental observations in most patients in whom a complete heart block appears the intact deeper centres immediately become active and prevent disturbances But if the block develops in a case in which the deeper centres are also injured by the same pathologic process which produces the block

cardia appear precisely in cases with auriculoventricular conduction disturbances This is conceivable because the same disease which produces the disturbance of conduction also may lead to irritative states in the specific tissues and thus to tachycardia Thus the ventricular fibrillation reproduced in Fig 171 arose in a patient who at the same time had heart block Many cases have been reported in which both types of attack have been observed in the same patient

But the diagnosis is not easy even if one has the opportunity of examining the patient *in* an attack The pulse is absent in both conditions The heart sounds also are inaudible in the tachycardial form as soon as a certain rate is exceeded On the other hand during ventricular standstill the regular working auricle can produce distinct sounds which lead one to imagine that the ventricle is active A tracing during an episode is desirable but since the attacks appear suddenly without warning this is not always possible except when they recur frequently In the latter instance one must apply the electrodes and wait until an attack occurs

From the standpoint of differential diagnosis epilepsy first comes under consideration If in the midst of health the patient suddenly falls down and becomes unconscious and convulsions appear this erroneous diagnosis is comprehensible The majority of cases observed by us came by way of the neurologic clinic Similar attacks also occur in advanced stenosis of the aortic valve Confusion with simple syncope is common

During the attack of Stokes Adams peculiar disturbances of breathing occur which consist of a severe dyspnœa followed by apnœa This dyspnœa is perhaps the result of an accumulation of abnormal metabolic products in the respiratory centre and the apnœa the result of a preceding hyperventilation in the dyspnœic stage If Stokes Adams attacks become frequent this respiratory disturbance may be confused with Cheyne Stokes breathing

Every attack is a serious and dangerous event Everything depends upon whether the blood is supplied to the brain before the centres are seriously damaged Every reawakening from a severe attack is a reawakening from death

Treatment is permissible only when it has been demonstrated whether the attack is produced by tachycardia or by a ventricular standstill Treatment without a definite diagnosis may cause serious injury The two forms require drugs which have exactly opposite actions If cardiac standstill exists stimulating remedies must be given which might be fatal in the tachycardias and in such cases are therefore to be strictly avoided On the other hand,

more rapidly owing to the insufficient blood supply to the *sinus* node

In Fig 228b ventricular automatism gradually occurs *again* The ventricular complexes show an altered form Only after several beats does the automatism reach its full rate

In this case it concerns a rare form of Stokes Adams in a patient already presenting heart block Temporarily the automatism of the ventricular centre fails The block appeared after coronary thrombosis

At all events it must always be assumed that two disturbances are necessary for this form of Stokes Adams to appear (1) the failure of auriculoventricular conduction, (2) the failure of ventricular automatism

In rare cases a transient cardiac standstill may also develop *through a vagal* inhibition that is neurogenically There are cases in which such extreme hyperexcitability exists in the receptors in the carotid sinus that the least pressure at the corresponding area of the neck produces standstill Such patients fall unconscious if the head is held in a certain position or if a stiff collar presses on the carotid sinus, cases are also known in which tumours or masses of glands press upon the carotid sinus and precipitate most severe attacks (Wenckebach and Winterberg Weiss and Ferris)

Likewise "vago vagal" reflexes originating in other places may induce cardiac depression For instance several cases of cardiac standstill due to swallowing have been observed in which a hyper sensitive area in the pharynx was found Cardiac standstill appeared if this was touched with a probe but if the area was anæsthetized with novocaine the attacks did not occur (Flaum and Klima) In the discussion of these attacks it must be mentioned that depressant reflexes originating in the carotid sinus may lead to disturbances of consciousness without cardiac standstill and without lowering of the blood pressure (Weiss and Ferris)

Clinical differentiation between the two forms of Stokes Adams (the form with tachycardia and that with cardiac standstill with its various subtypes) is not always easy The history of the patient is the same in the two forms One might believe that an attack of tachycardia would be assumed when extrasystolic allorhythmias are found during the examination of the patient in the interval between the attacks and that the variety with cardiac standstill is present when a disturbance of conduction exists outside the attack But often this does not hold in practice Thus experience of recent years has indicated that most attacks of circulatory standstill from tachy

it is especially effective in those patients whose attacks may be traced to coronary sclerosis

The administration of atropine has proved useful only when the attacks are caused by abnormally strong vagal reflexes and hyperexcitability of the vagus

Barium chloride has been recommended repeatedly for the treatment of cardiac standstill It has been known since the studies of Rothberger and Winterberg that the remedy increases the automatism of the deeper centres to an extraordinary degree If merely a few milligrams of $BaCl_2$ are injected intravenously in a dog ventricular fibrillation appears at once Cohn and Levine recommended very small doses by mouth in order to increase the automatism of the ventricle to the extent that ventricular standstill was prevented The dose amounts to 40 to 50 mg $BaCl_2$ which is administered three times a day (that is 20 drops of a 5 per cent solution three times daily) But this therapy has the disadvantage that in different people barium is absorbed from the intestine to a variable extent and at different rates moreover the borderline between the effective and toxic dose is very sharp and the margin of safety is small Thus at times no action is seen for a long time until suddenly a dangerous heterotopic tachycardia appears Opinions on the utility of barium are divided that of the authors being quite unfavourable The combination of barium with adrenalin-ephedrine preparations is likewise recommended by some authorities but because of the danger of ventricular fibrillation their use should be discouraged

Paradoxically if Stokes Adams attacks appear during the frequent transition from partial to complete block digitalis therapy often aids Conduction is still more impaired and soon a continuous complete block appears Then the automatism of the ventricle becomes continuously active and the attacks usually are no longer feared One administers small doses (0 2 gm (3 grs) of the standardized powdered leaf) of digitalis daily Since digitalis does not depress the automatism of the ventricular centres but promotes it this therapy is rational

Even operative treatment (section of one vagus) has been attempted and good results have been reported which perhaps should be ascribed to some other factor since the improvement occurred only several hours after the operation

One can hardly be too cautious in the appraisal of therapy, since the attacks very frequently cease quite soon even without treatment

the depressant agents effective in the tachycardias may cause serious damage if the attack is caused by cardiac standstill

The therapy of each case is divided into (1) the treatment of the attack itself, and (2) the prevention of new attacks

If the attack is produced by tachycardia, at first an attempt is made to abolish the tachycardia by one of the previously mentioned vagal reflexes (carotid sinus, bulbar reflex etc) If this is not successful or if a very high ventricular rate exists in auricular flutter or fibrillation where these reflexes are ineffective or only momentarily effective, it is best to give strophanthin (¼ mg) intravenously By this means the attack can be stopped (see p 351), or at least the ventricular rate can be reduced But it is clear that immediate aid is not provided with strophanthin since it is effective only after some minutes moreover it can reach the heart only when the circulation is still sufficient for this purpose As was stated earlier (p 388), this is often the case

It is well to avoid an intravenous injection of quinidine in these patients, since lowering of blood pressure and decrease of the contractile power of the heart may produce untoward effects But quinidine by mouth is always preferred when an attempt is made to prevent new attacks The dose under these circumstances would be as discussed in the chapter on tachycardias (p 351)

If a Stokes Adams attack is caused by ventricular standstill one must endeavour during the attack to stimulate ventricular automatism as much as possible A simple and very often effective measure consists in powerful blows in the cardiac region in animal experiments one succeeds easily in awakening automatism through mechanical stimuli

In standstill of the ventricles intramuscular or intravenous injections are useless The intracardiac injection of epinephrine is dangerous because too marked irritation of the centres may lead to the appearance of ventricular tachycardia and even fibrillation A heart in which a block has developed tends to form heterotopic stimuli Ventricular extrasystoles are found with remarkable regularity in these cases Therefore caffeine is recommended for intracardiac injection rather than epinephrine

But if the appearance of new attacks is to be prevented the conduction to the ventricle and ventricular automatism must be improved For this purpose preparations of epinephrine or ephedrine are useful One administers ephedrine by injection or tablets (0 03 gm) three times a day, theophylline (aminophylline) is also very effective as a vasodilating agent that increases automatism

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In the most common form of A-V rhythm the auricles and ventricles contract simultaneously the excitation which arises in the middle of the A-V node is conducted backward to the auricles and in the normal way to the ventricles Owing to the very poor conduction in the A-V node it is possible for the excitation to require just as much time for the shorter passage to the auricles as for the longer journey to the ventricles As demonstrated in Fig 229b

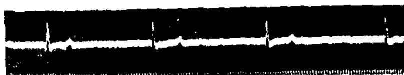


Fig 230 Auriculo ventricular rhythm (middle section of node)

the auricles and ventricles are stimulated simultaneously and the P waves which appear at the same time as the initial deflections are concealed in them and therefore are invisible

Fig 230 shows in Lead II a tracing of this type A slow ventricular rhythm was seen in all leads P waves were absent They were also absent when a chest lead was employed (see p 32) in other words their invisibility does not depend simply upon their small amplitude In animal experiments this form of A-V rhythm may be observed directly and the auricles are seen to contract simultaneously with the ventricles



Fig 231 (Lead II) Auriculo ventricular rhythm (upper section of node)

If the stimulus arises high in the A-V node it passes back to the auricles very rapidly and the path to the ventricles requires a somewhat longer time as is indicated in Fig 229a Owing to this type of spread of the excitation rhythmic cardiac action is seen in which the auricles are stimulated shortly before the ventricles and the P-R interval is short Since the direction of auricular excitation is directly opposite to the normal the P waves are negative in Leads II and III They are low positive in Lead I *This form of A-V rhythm is recognized by the fact that negative P waves are followed at a short interval by normal ventricular complexes*

XXIII

AURICULOVENTRICULAR RHYTHMS AND ARRHYTHMIAS

MANIFESTATIONS OF AURICULOVENTRICULAR RHYTHM

WHEN the sinus node loses its activity, or by virtue of a disturbance of sino auricular conduction, the stimuli formed there are not conducted the centres located in the A–V node (" secondary centres ") assume control over the ventricle

The centres of the A–V node possess a well developed automatism *Their rate is only slightly less than that of the sinus node,* so that in the event of an A–V rhythm there is only a slight reduction of cardiac rate The patient scarcely ever comes to the physician because of a pronounced bradycardia

The A–V node is a poor conductor A stimulus conducted from

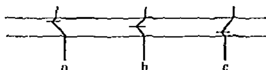


FIG 229 Schematic drawing of the three forms of auriculoventricular rhythm

the auricle to the ventricle is held up for approximately 0 05 second in the A–V node so that the auriculoventricular conduction time normally has the remarkable duration of at least 0 12 second This has a definite practical significance because it gives the auricle sufficient time to force blood into the ventricle to augment its filling and increase the tension of its musculature

According to the electrocardiographic picture three forms of A–V rhythm are distinguished, and a different site of origin in the *A–V node is assumed for each one Against the justification of this* localization there are a number of facts based upon experimental observations (Scherf) that it is not proposed to consider in this work The concealed position of the A–V node in the heart makes an experimental study of its behaviour in conduction and stimulus formation very difficult At present our knowledge of the physiology of the A–V node is very meagre

As long as results of further investigations are not available the customary explanation of the three forms of A–V rhythm will be retained

In the most common form of A-V rhythm the auricles and ventricles contract simultaneously the excitation which arises in the middle of the A-V node is conducted backward to the auricles and in the normal way to the ventricles Owing to the very poor conduction in the A-V node it is possible for the excitation to require just as much time for the shorter passage to the auricles as for the longer journey to the ventricles As demonstrated in Fig 229b

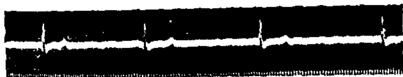


FIG 30 Auriculo ventricular rhythm (middle section of node)

the auricles and ventricles are stimulated simultaneously and the P waves which appear at the same time as the initial deflections are concealed in them and therefore are invisible

Fig 230 shows in Lead II a tracing of this type A slow ventricular rhythm was seen in all leads P waves were absent They were also absent when a chest lead was employed (see p 32) in other words their invisibility does not depend simply upon their small amplitude In animal experiments this form of A-V rhythm may be observed directly and the auricles are seen to contract *simultaneously* with the ventricles



FIG 31 (Lead II) Auriculo ventricular rhythm (upper section of node)

If the stimulus arises high in the A-V node it passes back to the auricles very rapidly and the path to the ventricles requires a somewhat longer time as is indicated in Fig 229a Owing to this type of spread of the excitation rhythmic cardiac action is seen in which the auricles are stimulated shortly before the ventricles and the P-R interval is short Since the direction of auricular excitation is directly opposite to the normal the P waves are negative in Leads II and III They are low positive in Lead I *This form of A-V rhythm is recognized by the fact that negative P waves are followed at a shorter interval by normal ventricular complexes*

An example of this form of A–V rhythm is reproduced in Fig 231 The cardiac rate is somewhat accelerated (rate 95) Negative P waves are visible before the ventricular complexes which are normal in configuration The conduction time amounts to only 0 08 second

Recently it was demonstrated on dogs that origin of the stimulus in the upper part of the auriculoventricular node around the opening of the coronary vein actually leads to a rhythm with inverted P waves and a normal or almost normal P–R interval (Scherf and Harris) This type of rhythm is also called "coronary sinus rhythm" The P waves are usually low and positive in Lead I and inverted in

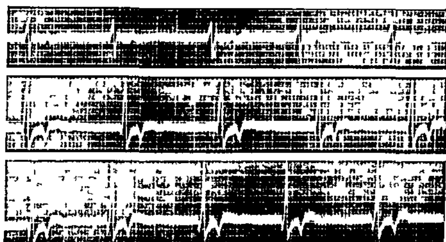


Fig 232 Auriculoventricular rhythm (lower section of node)

Leads II and III It has not been decided whether this rhythm can be separated from "upper" A–V rhythm

If the stimulus arises in the lower part of the A–V node it reaches the ventricles very rapidly and the conduction back to the auricles through a large part of the A–V node requires a longer time The ventricles as indicated in Fig 229c are activated earlier than the auricles the QRS complexes appear first and the P waves follow Usually a negative P wave is found between the QRS and the T waves

In Fig 232 the cardiac rate is regular No P waves are seen before the ventricular complexes In Lead I slight notching of the T wave is visible and it cannot be definitely determined whether or not a P wave is present In Leads II and III however deep negative P waves are distinctly recognizable in the RS–T interval

Tracings showing the latter two varieties of A–V rhythm (origin

of the stimulus in the upper and lowermost sections of the A-V node) are uncommon

An A-V rhythm can very easily be produced in the healthy individual under two conditions First, it is found commonly during a carotid pressure test (see Fig 205) During vagal inhibition the sinus node loses its activity while the A-V node which is much less subject to vagal influence continues to work Second it is found after the administration of atropine (Wilson) About 30 per cent of normal people transiently show A-V rhythm soon after a sub cutaneous or intravenous injection of atropine has taken effect (Eckl) Atropine produces an inverse excitation of the vagus which is evident for a short time before the paralytic effect becomes apparent Apart from this atropine does not paralyse all vagal endings in the heart simultaneously Thus it may happen that for a short time the A-V nodal centres function more rapidly than the centres in the sinus node Since the more rapid rhythm controls the heart an A-V rhythm is found for the duration of this state In these artificially induced A-V rhythms almost always that variety is present in which the auricles and ventricles are stimulated simultaneously and the P waves are hidden in the initial deflections

Parade found a transient A-V rhythm after a cold bath he ascribed this phenomenon to variations in the tonus of the vegetative nerves

An A-V rhythm may be caused in two ways fundamentally different in their modes of origin first (as already mentioned) *when no stimuli pass from the sinus node to the auricle that is in* an incomplete or complete sinus block (in this instance the A-V rhythm is a sequela of a disease *in the region of the sinus node*) second when the automatism of the A-V node is higher than that of the sinus node If the cardiac rate is slow (as in Figs 230 and 232) the possibility mentioned first is assumed but if the rate is high (as in Fig 231) the second comes under consideration

A-V rhythm is frequently referred to as nodal rhythm It is advisable to avoid this designation since sinus rhythm is also a nodal rhythm moreover some time ago fibrillation often was mistakenly called nodal rhythm (Mackenzie) and this name still appears in older books and adds to confusion

In two forms of A-V rhythm the auricles and ventricles contract *more or less* simultaneously and the auricles cannot empty their contents into the ventricles the sphincter mechanism at the mouths of the *great veins which normally* prevents regurgitation is over come and the blood is expelled backward into the large veins Thus in A-V rhythm high superimposed waves are at times seen

in the neck Likewise in A–V rhythm the first heart sound is often strikingly accentuated

No symptoms are produced by an A–V rhythm, the appearance of venous pulsations in the neck is perceived by only a few patients The abnormal mechanism produces no enduring circulatory damage since the cardiac rate, as a rule, is sufficiently slow to permit the ventricles to fill during the long diastoles without the aid of the auricles



Fig 233 (Lead II) Interference between sinus and auriculoventricular rhythm

INTERFERENCE OF SINUS AND AURICULO-VENTRICULAR RHYTHMS

If the sinus and A–V centres form stimuli with *approximately equal rates and if both* centres show slight but not completely parallel *rhythmic variations very confusing tracings* may appear

At times a normal P wave is observed at a normal interval *before* the ventricular complex and a sinus beat must be assumed at times a negative P wave is seen at a normal or shorter interval before or after the initial deflection A change in the site of origin of the stimulus a shifting of the pacemaker appears

It should be recalled that sinus rhythm may exist even if negative P waves appear at *normal* intervals before the initial deflection the spread of excitation in the auricles being abnormal this may result from an origin of the stimulus in the lower end of the sinus node (Rothberger and Scherf)

In Fig 233 varying beats may be observed with normal and negative P waves which are located about 0 15 second before the initial deflection but repeatedly ventricular complexes appear which are neither preceded nor followed by a P wave that is where the P wave is concealed in the initial deflection (see also Fig 236)

INTERFERENCE DISSOCIATION

In the discussion of ventricular extrasystoles reference was made to the fact that often they are not transmitted back *to the auricle*

since they are held up in the A-V node which conducts poorly even in a normal direction This depression of retrograde conduction also occurs sometimes in A-V rhythm mainly in that form which develops through increased automatism of an A-V centre (that is, in the form which is accompanied by a higher rate) At times it is observed in the course of digitalis therapy Usually it is transient

Since the A-V stimuli are not conducted back to the auricle the sinus node works undisturbed

When the A-V centre is functioning more rapidly than the sinus node centre (otherwise its action could not become manifest) it forms independent stimuli interfering with those of the sinus node Every sinus beat which occurs outside of the refractory phase of an A-V beat is conducted to the ventricle since the auriculoventricular conduction is normal The conducted beat breaks into the A-V



FIG 34 (chest lead) Interference between sinus and auriculoventricular rhythm (interference dissociation)

centre so that its stimulus formation begins anew and a ' coupling of two rhythms appears According to the rate relationships of the two rhythms interfering with one another the most diverse pictures develop

Tracings of this type have been known for a long time They were correctly interpreted for the first time by P D White and Wilson and were named interference dissociation by Mobitz

In Fig 234 which was obtained from a strongly digitalized patient who suffered from a mitral lesion an interference dissociation may be seen The sinus rhythm which was depressed owing to the action of digitalis upon the vagus had a rate of 41 the A-V rhythm a higher rate of 48 Since the A-V beats were not conducted back to the auricle interference of the two rhythms occurred At the beginning of the tracing an automatic beat may be seen in whose RS-T interval a P wave is concealed immediately after the initial deflection Then an automatic (A-V) beat occurs the normal

Fig 235 Interference dissociation

P wave which immediately follows on this occasion *appears so late that it is conducted to* the ventricle although somewhat slowly Then the interference begins again The next normal P wave is concealed in the initial deflection of the A–V beat, the following P waves come increasingly later until one occurs so late that the stimulus is again conducted to the ventricle

A similar tracing is seen in Fig 235, in this case both centres (the sinus node as well as the A–V node) work more rapidly and the difference in rate of the two rhythms is very slight so that shifting occurs more slowly This tracing was recorded from a digitalized patient (hypertension and coronary sclerosis) in *both cases the* disturbance vanished after the discontinuance of digitalis therapy The third and the last but one ventricular complexes in Fig 235 are conducted from the auricle, the *other complexes* originate in the A–V node Clinically one obtains the impression that extrasystoles exist The analysis of the tracings proves, however, that the extrasystoles are in reality normal sinus beats

Interference dissociation is rare in non digitalized patients In the absence of digitalis it occurs in disturbances of the sinus node when the sinus rhythm is sufficiently slowed as to enable an A–V rhythm to appear The failure of conduction of the stimulus from the A–V node back to the auricle is always a basic condition *for the appearance of the arrhythmia* At times an interference dissociation is found in a 2 1 sinus block because in this instance the A–V node may work more rapidly than the sinus node

In experimental work an interference dissociation is readily produced by aconitine

If the sinus and A–V rhythms have almost the same rate, but show very slight variations and if retrograde conduction from the A–V node to the auricle is continually *absent tracings like* Fig 236 develop

Between Figs 236a and 236b a section of the tracing containing three ventricular complexes has been removed because they displayed the same form as those at the end of Fig 236a or the beginning of Fig 236b At times the P waves of the sinus rhythm appear before at times after the QRS complexes and at times they are concealed in them The P wave is continually positive, that is the auricle is regularly stimulated by the sinus node An A-V centre forms stimuli for the ventricle (competition between two centres) (p 402)

With marked differences of rate between the sinus and A-V rhythm tracings appear (see Figs 226 and 227) which it is advisable to consider separately from actual interference dissociation with slow shifting of two rhythms though no fundamental difference exists

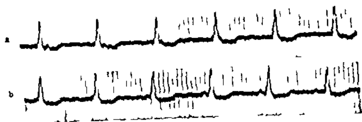


Fig 236 Competition between sinus and auriculoventricular nodes

In rare cases a centre in the sinus node interferes not with the A-V centre but with a centre of stimulus formation lying deeper in the ventricle which works more rapidly than the sinus node As in Fig 237 the ventricular complexes of the automatic ventricular beats are abnormal in shape In Fig 237 the sinus node forms 85 stimuli while the ventricular centre yields 90 stimuli per minute Here also the ventricles respond to each stimulus from both centres as long as they occur outside the refractory period The eighth QRS complex in Fig 237 is conducted from the auricle the ninth shows some changes because a part of the ventricle is stimulated by the sinus stimulus and another part by the idioventricular stimulus (summation beat)

AURICULOVENTRICULAR EXTRASYSTOLES AND TACHYCARDIAS

Since (as was stated on p 241) every specific fibre of the heart is able to form automatic stimuli and may also be the site of origin of pathologically formed stimuli extrasystoles and tachycardias may also arise in the A-V node

Fig 238 shows extrasystoles occurring between several normal beats, superficial inspection might cause one to diagnose auricular extrasystoles Closer examination, however, reveals that the auri-



Fig 237 Interference between sinus node and a deep ventricular centre



Fig 238 (*Lead II*) Auriculoventricular extrasystoles originating in upper part of A-V node

culoventricular conduction of normal beats amounts to 0 15 second but the P waves appearing prematurely have a P–R interval of only 0 11 second In the discussion of auricular extrasystoles it was stressed that, owing to their prematurity, they are often conducted slowly to the ventricle, so that a prolongation of auriculo

ventricular conduction time appears But since in Fig 238 premature *negative* auricular waves are followed by ventricular complexes of normal appearance at a shortened interval one must assume the presence of auriculoventricular extrasystoles whose origin is in the upper section of the A-V node



Fig 239 Auriculoventricular tachycardia originating in upper section of A-V node

Fig 239 shows a short segment of a record of paroxysmal tachycardia in which inverted P waves precede the ventricular complexes by very short periods During the sinus rhythm between the attacks conduction time was almost twice as long In other words, an A-V tachycardia originating from the upper part of the A-V node is present

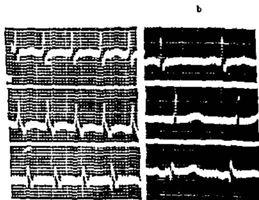


Fig 240 Paroxysmal tachycardia originating in lower part of A-V node (a) and after its disappearance (b)

Fig 240a was obtained during an attack of paroxysmal tachycardia which lasted three and one half hours and which was abolished by an intravenous injection of 0 25 mg of strophanthin Fig 240b shows the electrocardiogram from the same patient immediately after the disappearance of the tachycardia During the attack inverted waves are visible especially in Leads II and III, between

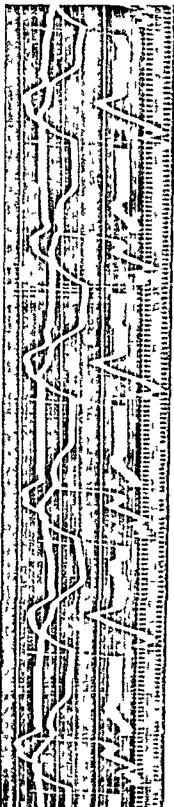


Fig 241 Dog experiment A-V rhythm originating in lower part of A-V node with delayed reversed conduction to auricle of every second stimulus whenever reversed conduction is prolonged the stimulus returns from auricle to ventricle (return extrasystole or reciprocating beat) Time marker 1/50th second

the QRS complex and the T wave These waves are not found in Fig 240b, we may assume that they are P waves and therefore that a tachycardia originating from the lower part of the A-V node exists

RECIPROCATING BEATS (RHYTHM)

Under certain experimental conditions in the frog and fish heart, Mines discovered an interesting disturbance of rhythm Auricle and ventricle contracted alternately and it could be shown that a circulating wave existed, *the same stimulus* being conducted from the auricle to the ventricle and again back to the auricle etc Since a broad muscular communication exists between the auricle and ventricle in fish and amphibian hearts it might easily happen that a stimulus employs one part of the tissue uniting the auricles and ventricles for the ventriculo auricular conduction and *another* part for the auriculoventricular conduction This is one of the conditions in which the existence of a circus movement is proved and it was one of the standard experiments upon which Mines based his belief that auricular fibrillation might be caused by a continuous circus movement

It is of great interest that dis *turbances produced by a similar* mechanism were discovered in man originally by P D White and

A V Drury The broad communication between auricle and ventricle is reduced in man to a small path and such a circulating wave would not be expected *a priori* There is no doubt however that such a mechanism exists and a few additional cases have been described in recent years Usually in these cases that form of A-V rhythm is present in which the ventricles contract before the auricles and an inverted P wave is visible between the S and T waves If the reversed conduction from the node to the auricle is delayed (as the result of digitalis therapy or carotid sinus pressure) the P wave appears later after the QRS complex and it is again followed by a ventricular complex

Fig 241 was obtained in a dog experiment (Scl erf) The upper tracing shows the mechanogram registered from the right auricle the second tracing is a mechanogram from the right ventricle the electrocardiogram follows and finally the time marker which indicates 1/o0th of a second An A-V rhythm exists and the inverted P wave is visible immediately after the QRS complex

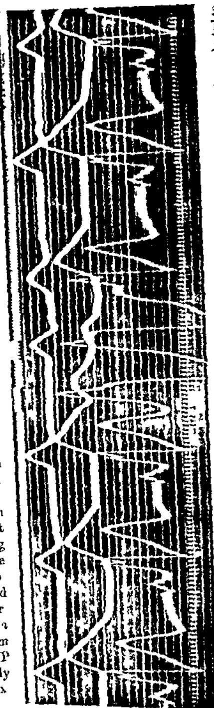


Fig 241 Dog experiment [illegible] ventricular extrasystoles [illegible] conducted to auricle and [illegible] returns immediately to ventricle [illegible] Time marker 1/50th [illegible]

There is also alternans of reversed conduction since every *second* complex shows the P wave farther after the initial complex Every time the P wave appeared later it was followed by a second QRS complex The stimulus which had just been conducted to the auricle turned back and again activated the ventricle According to the law of the absolute refractory phase, the stimulus could not be conducted over the same path twice in such rapid succession Therefore a functional dissociation of the upper part of the auriculoventricular conduction must be postulated, one part of it being employed for the upward another part for the downward conduction Schmitt and Erlanger found similar phenomena in small strips of muscle from the turtle s heart

Ventricular extrasystoles in dogs are frequently conducted backward to the auricle If the reversed conduction is slightly delayed, the same 'return extrasystole' may appear (Scherf and Shookhoff)

Fig 242 was obtained in a dog experiment After three sinus beats two ventricular extrasystoles appear, induced by induction shocks (the upper tracing indicates the opening and closing shock) The second ventricular extrasystole is conducted backward to the auricle since a premature contraction appears there, this is indicated in the second tracing which registered mechanically the contractions of the right auricle The backward conducted second ventricular extrasystole returns from the auricle to the ventricle (return extrasystole, reciprocating beat) The dog received 0 4 gm quinine hydrochloride intravenously before the experiment therefore the QRS complexes are widened

This is a type of ventricular extrasystole for which a re entry mechanism is proved It is possible that similar phenomena in the peripheral parts of the specific tissue within the ventricles are the cause of other extrasystoles The role played by the phenomenon of supernormal phase of conductivity in this re entry mechanism is not well known at present

Abnormally short P–R intervals (less than 0 12 second) with sinus rhythm and positive P waves are occasionally observed in the born born heart and in certain types of hypertension with marked hypermotility of the heart and a rapidly progressive course (Scherf)

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XXIV

PARASYSTOLE

In the discussion of extrasystoles (p 241) it was emphasized that every specific fibre has the ability to form rhythmic stimuli The fibres lying in the sinus node have *the most* highly developed automatism and the other centres work *the more slowly* the nearer they are to the cardiac apex In the normal heart every stimulus originating in the sinus node on its way over *the heart breaks into the* deeper centres and there destroys all material in the process of stimulus formation so that the automatism of *the deeper centres cannot appear*

Under abnormal conditions this behaviour can be disturbed A deeper centre may be altered in such a way *that it no longer* responds to a conducted stimulus perhaps because its irritability is lowered or because the stimulus conducted is weak In this case stimulus formation proceeds undisturbed in this centre which usually lies in the ventricle

Under these circumstances there are two sites of stimulus formation in the heart first the normal pacemaker *in the sinus node* second an automatic centre forming undisturbed stimuli All stimuli formed by these centres which fall outside *the refractory* period yield a response In a tracing from such a case a regular sinus rhythm and a regular automatic rhythm will be found working independently of each other Thus it happens that the ventricles may occasionally be stimulated by both sinus and automatic stimuli at the same moment because of the continuous interference of both rhythms the interval between an automatic beat and the preceding sinus beat continuously varies In contrast to interference dissociation where the two rhythms are coupled with each other every time a sinus beat is conducted to the ventricles here *the two rhythms work entirely undisturbed and completely independent of each other* as in heart block Kaufmann and Rothberger to whom we owe the discovery and investigation of these disturbances of normal cardiac rhythm introduced the names pararrhythmia and parasystole since a second independent rhythm prevails in the heart *besides* (para) the normal rhythm

In Fig 243 superficial inspection suggests the presence of sinus rhythm which is disturbed by isolated extrasystoles But closer inspection reveals that the time of occurrence of the beats suggestive of extrasystoles varies constantly with those beats at times coming early occasionally late in diastole their coupling or interval



Fig 243 Para systole with simple interference of two centres

after the preceding normal beat varies Measurement demonstrates that the interval between the abnormal beats is always a multiple of the interval between such abnormal beats which follow each other directly we have, therefore to conclude that a centre works rhythmically in the ventricle, undisturbed by the sinus rhythm One finds that some abnormal beats appear more or less simultaneously with sinus beats so that the ventricular complexes show every transition between the two main forms sometimes a larger part of the ventricle is excited by abnormal automatic stimuli, at times by the sinus stimulus so that mixed forms (summation) of the ventricular complexes appear The sinus rhythm has a cycle length of 0 65 second (rate = 92) The automatic ventricular stimuli have a cycle length of 1 22 to 1 24 seconds (rate = 48 to 49) The interval in the middle of the tracing that is free from abnormal beats has a length of 4 85 seconds that is it corresponds to four times the automatic period If over a period of weeks long sections of tracings are recorded daily the same situation is always present *Measurement always demonstrates that every automatic stimulus which appears when the ventricle is no longer in a refractory stage after a sinus beat yields a ventricular response*

Indeed according to the relation between the rates of the two rhythms pictures of the most diverse types of interference appear they can be easily predicted by reconstruction on graph paper

Parasystoles are not rare when one investigates every tracing showing extrasystoles ' which appear in varying phases of diastole in order to see whether or not an automatic rhythm, independent of the sinus rhythm interferes with the latter Actually in para systole we are not dealing with extrasystoles because the latter do not develop independently by an auto matic stimulus formation but (as was mentioned on pp 268 and 272) are caused by the preceding beat Therefore the interval between the extrasystole and the preceding precipitating beat in a given case is always the same In parasystole on the other hand the interval between the two types of beat varies

The chief factor in the appearance of parasystole is the undisturbed operation of an automatic centre which is isolated by a protective block (Kaufmann and Rothberger) from other centres of stimulus formation as was stated previously this does not respond to outside stimuli and is not disturbed by them The presence of such a protective block has been experimentally proved (Scherf) Actually it is not a block in the sense understood for many years but an inability of the ventricular centre to respond to outside stimuli

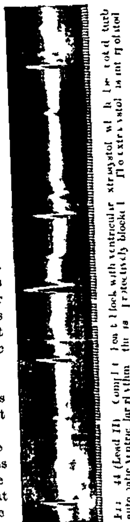


Fig 244 (Lead II) Complete heart block with ventricular extrasystole which does not disturb automatic ventricular rhythm the is protectively blocked The extrasystole is interpolated

A clear example of a protective block is furnished by Fig 244 Here a complete heart block is present since almost regular auricular waves and regular ventricular complexes appear and the auriculoventricular intervals vary continually After the second ventricular beat a ventricular extrasystole is seen which appeared during carotid pressure (the delicate white line shortly in front of the extrasystole is the signal and indicates approximately the beginning of carotid pressure) As a rule a ventricular extrasystole in heart block disturbs the activity of the automatic ventricular centre since the extrasystolic stimulus breaks into the automatic centre so that it must begin its work anew and the post extrasystolic pause is just as long as an automatic period In Fig 244 this is not the case The automatic centre works undisturbed and its rhythm is not changed by the extrasystole it was protectively blocked

As a rule the rate of an automatic centre corresponding to its position in the heart is much slower than that of the sinus node But at times its rate may be higher than that of the sinus rhythm of the affected case Since the more rapid centre always controls the heart a ventricular tachycardia should now appear But it may happen that some of the rapidly formed ventricular stimuli elicit no response from the ventricle or are not further conducted (exit block) so that during the ensuing pause sinus stimuli immediately step in Then pictures like Fig 245 may be seen

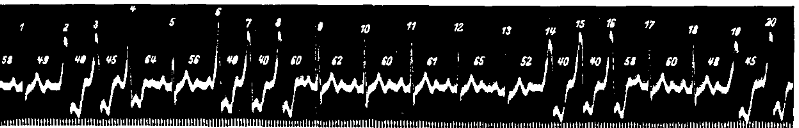


Fig 245 Parasystole with exit blockade



Fig 246 Paroxysmal ventricular tachycardia some extrasystoles are missing through exit block

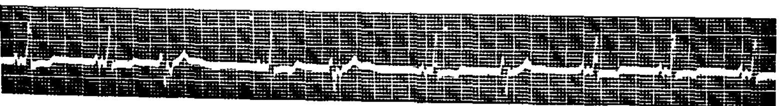


Fig 247 Parasystole with simple interference of two rhythms and reversed conduction of the first two ectopic ventricular beats to the auricle (Lead II)

Here a sinus rhythm with a rate of 92 to 100 is present It is interrupted frequently by groups of two or three abnormal ventricular beats which look like extrasystoles Repeatedly mixed (summation) complexes appear The time of occurrence of abnormal beats varies this is against an extrasystolic mechanism and in favour of increased automatism The cycle length of the automatic beats amounts to 0 40 to 0 45 (mostly 0 40) second and the rate is approximately 150 The three intervals depicted which are filled by sinus beats between the abnormal beats amount to 1 20 (3 × 40) 3 40 (9 × 40) and 1 64 (4 × 41) seconds Since in long records this irregularity was always found in the same way the existence of a rhythmically active abnormal ventricular centre must be assumed in this case But the measurement and calculation of the tracing shows that in contrast to Fig 243 not every stimulus formed by the abnormal centre and actually appearing outside the refractory phase of the ventricle elicits a response from the ventricle

A good example of exit block without parasystole is furnished by Fig 246 It was recorded in a case which is repeatedly mentioned in the book of Wenckebach and Winterberg (observation of Scherf Winterberg) It concerns a paroxysmal ventricular tachycardia In Fig 246 a new attack begins after a normal sinus beat twice the chain of extrasystoles is interrupted The pause thereby developing *is exactly twice as long as an extrasystolic period* The extrasystoles appear at intervals of 0 45 to 0 48 second the two omission periods measure 0 90 to 0 92 second In other words there is an exit block and twice the ventricle does not respond to the extra stimulus

The rare form of parasystole which is shown in Fig 245 is called parasystole with *protective and exit block* Sufficient explanation of the appearance of both forms of parasystole (with and without exit block) is found simply in the relation between the strength of the normally conducted stimulus or the automatic stimulus on the one hand and the irritability of the tissue surrounding the centre or of the centre itself on the other It is *unnecessary* to assume an imaginary block line surrounding the abnormal centre in the ventricle as did Kaufmann and Rothberger

Fig 247 shows a parasystole with simple interference of two rhythms and reversed conduction of the first two ectopic ventricular beats to the auricle

It is interesting that all parasystoles reported up to the present as far as we know have been found exclusively in patients who have heart disease or in whom the history or the clinical findings permit one to suspect it (Faltitschek and Scherf)

XXV

THE PRE-EXCITATION SYNDROME

In lower animals the auricles are connected by bands of muscle with the ventricles along the entire A–V junction In mammals this extensive connection is reduced to the small atrioventricular conduction system

The primitive anlage of the heart in mammals represents a "cardiac tube" around which a muscular mantle develops The cardiac tube is the earliest stage in the development of the specific tissue, and the superimposed muscle fibres then form the common muscle Here, as elsewhere in the heart, *ontogeny recapitulates* phylogeny

In the course of investigations which were designed to determine how the continuity of the embryonic cardiac tube is interrupted *during its development, the anatomist* Kent *(1892) found in recently* littered rats, a muscular connection between the right auricle and ventricle Kent was able to demonstrate experimentally the capacity of this bundle for conduction If he sectioned all the connecting tissue between the auricles and ventricles with the exception of the right lateral connection co ordination between the auricles and ventricles remained (Paladino described similar connecting fibres between auricle and ventricle)

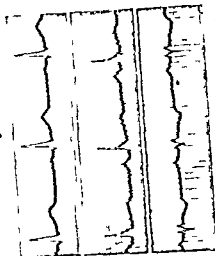
This discovery was soon re examined and confirmed however since the discovery of the bundle of His, the bundle of Kent has fallen into oblivion This is comprehensible in view of the pre eminent significance of the auriculo ventricular connection in representing the A–V conduction system

In recent years however peculiar electrocardiograms have been reported (Wolff Parkinson and White) which at first were interpreted otherwise but according to our view and on the basis of present knowledge they can be explained without difficulty by the assumption of an accessory A–V bundle (Holzmann and Scherf Scherf and Schönbrunner Wolferth and Wood)

Fig 248 was obtained from a forty six year-old patient who claimed that he had always been healthy In recent years he had complained of attacks of palpitation which at first appeared only after exertion and excitement but subsequently without any apparent cause They lasted from one half hour to two hours and began and ended suddenly, in the attacks the pulse was irregular Clinical examination revealed an entirely normal cardiac status

The electrocardiogram was abnormal (Fig 248a) There was a regular cardiac action with a conduction time of 0 11 second and the initial deflections were notched and 0 12 second in width the T waves were also abnormal After knee bending exercises a normal electrocardiogram with a conduction time of 0 17 second could always be obtained (Fig 248b) Moreover the two types frequently alternated with each other spontaneously

A forty four year-old woman reported that for more than twenty two years she had suffered from attacks of palpitation which the electrocardiogram proved to consist of paroxysmal auricular tachycardia In this patient likewise the cardiac examination was normal She also showed at times abnormal and at times normal electrocardiograms



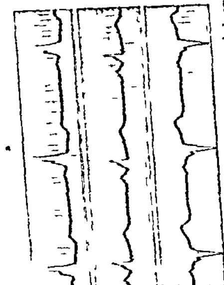


Fig 248 (a) Shortened conduction time and abnormal ventricular complexes (b) Normal electrocardiogram recorded after some knee bending exercises

In Fig 249 there is a spontaneous change between the two types Despite a constant sinus rhythm and constant auricular activity abnormal ventricular complexes repeatedly occur after normal P waves Moreover in these cases the initial deflections are wide and the terminal deflections (especially in Leads II and III) are abnormal

On the basis of analysis of these cases it became evident that

with constant auricular activity, occasionally or for longer periods, abnormal ventricular beats appeared *which were always dependent upon the auricles,* and which have a shortened conduction time

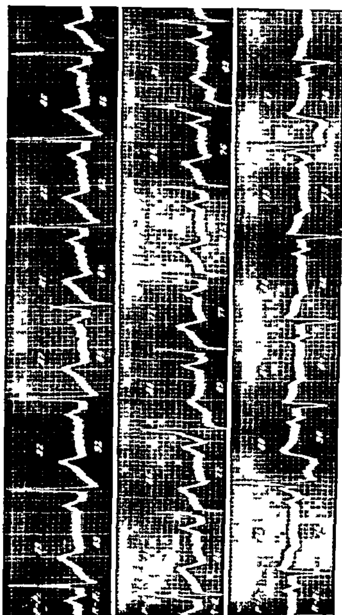


Fig 249 Interchange of normal and abnormally conducted beats

(usually under 0 12 second) it was therefore assumed that in addition to the A–V conduction system a second auriculo ventricular path of conduction existed in such cases The location of this abnormal pathway of conduction seems to vary But since in all

known cases of this type after a shortened conduction time *abnormal* ventricular complexes always appear indicating a disturbance of intraventricular conduction it was assumed that this second path reaches the ventricle at an abnormal site The duration of con duction in the abnormal path is shorter mainly because the delay of con duction in the A–V node is absent The conducted auricular stimulus can at times employ the normal at times the abnormal path (Fig 249) In one patient the change may take place after a couple of beats in another after months or years Sometimes only abnormal beats are observed Very often one succeeds by means of an exercise test by atropine or amyl nitrite in converting one form into the other Alterations of irritability of the tone of the cardiac nerve result in the auricular stimulus employing the path in which it finds the least resistance

Fig 250a shows alternating normal and abnormal beats About half way in 250a three abnormal beats follow each other In 250b which was obtained from the same patient a few

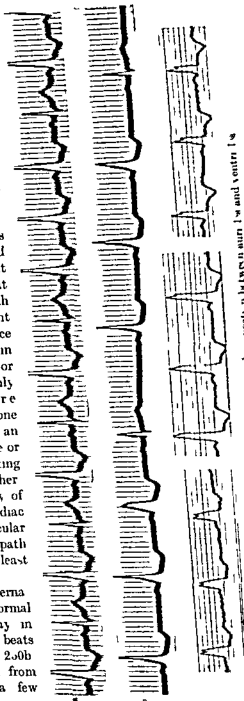


Fig 250 Three instances of abnormal connection between auricles and ventricles

with constant auricular activity, occasionally or for longer periods abnormal ventricular beats appeared *which were always dependent upon the auricles,* and which have a shortened conduction time

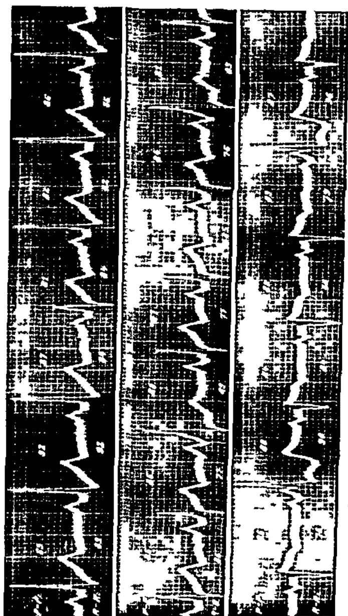


Fig 249 Interchange of normal and abnormally conducted beats

(usually under 0 12 second) it was therefore assumed that in addition to the *A–V* conduction system a second auriculo ventricular path of conduction existed in such cases The location of this abnormal pathway of conduction seems to vary But since in all

at first glance they may seem abnormal (as in Figs 248-256) or may reveal an approximately normal appearance (as in Fig 251) and thus they are easily misinterpreted Also the direction of the deflection in Leads I and III can be different However the notch near the base of the ascending limb of the R wave is found very frequently

It is interesting that the majority of patients with abnormal auriculo ventricular connection consult the physician because of attacks of paroxysmal tachycardia Thus twenty of thirty-one patients with cases of this type complained of sudden palpitation

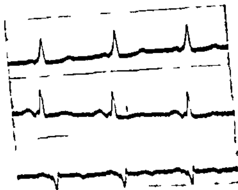


Fig 1 Shortened conduction and abnormal initial deflection in patient with attacks of paroxysmal tachycardia.

The relationship is not quite clear However it may be recalled that many years ago before cases of this kind were known de Boer elaborated a hypothesis of the development of paroxysmal tachycardia which was based upon the assumption of the existence of the bundle of Kent According to his theory paroxysmal tachycardias develop from a continuous circulation of a wave of excitation His theory presumes that the excitation in auricular tachycardia uses the following path auricle bundle of His ventricle bundle of Kent auricle If the excitation proceeds in a reverse direction (i e down the bundle of Kent and up the bundle of His) then ventricular tachycardia was supposed to occur Some doubt may be expressed regarding this theory (see Fig 268) and the question of the cause of paroxysmal tachycardia (auricular or ventricular tachycardia fibrillation or flutter) in these cases still remains unanswered (see Fig 268)

According to Scherf a ventricular contraction conducted reversedly *via* the anomalous pathway may reach the auricle during

minutes after an exercise test a bradycardia with marked sinus arrhythmia is recorded Two normal ventricular complexes appear At two places the abnormally conducted beats show a particularly marked widening (immediately before the sinus beats with *normal* conduction) It is probable that both pathways that over the A-V conduction system and over the anomalous path are used, the form of the QRS complexes is the more abnormal the more the ventricles are activated over the anomalous path Fig 250c also shows abnormal ventricular beats following a few hundredths of a second after *normal* P waves The initial deflections in this case are especially wide and closely resemble the tracings obtained in left *bundle* branch block

Both cases were normal in regard to the clinical examination of the circulation In each instance it concerned patients who sought relief for a paroxysmal tachycardia

Since the condition is due to a harmless congenital anomaly (the youngest patient observed was four and a half years old) the prognosis is good, the patients are usually healthy from a cardiac standpoint and may not be aware of their anomaly until old age

Recently Glomset and Glomset discovered muscular bridges between the auricle and ventricle in the auriculo ventricular groove in the dog and human quite independent of the bundle of His

Three muscular connections at the right lateral border between auricle and ventricle were demonstrated recently in a case with a shortened P–R interval and abnormal ventricular complexes (Wood Wolferth and Geckeler)

From an embryologic point of view it is indeed possible that at times other remnants of the original extensive connection normally found in the primitive heart may persist which are capable of conduction

In recent years other explanations have been offered for this syndrome (references in Oehnell s work) but most of these are not well founded One possibility which is often discussed has been considered but rejected by Holzmann and Scherf that is the existence of an irritable focus in the ventricle which forms an extra systole whenever the auricle contracts

Knowledge of this anomaly is important since the physician often overlooks the shortened conduction time and deduces the existence of a myocardial disease from the abnormal ventricular complexes Repeatedly we have seen such cases unnecessarily treated as myocardial disease

The ventricular complexes may show quite different pictures

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the so called vulnerable phase of diastole when *one* stimulus suffices to cause a tachycardia or fibrillation The same may hold *vice versa* for ventricular tachycardias in this syndrome

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4 Abnormal T waves in Leads I and II (digitalis!)
5 Uniform ventricular extrasystoles
6 All forms of conduction disturbances (when the administration of digitalis can be excluded)
7 Unequivocally abnormal result of the exercise test

As frequent and important signs of a myocardial disease but not absolutely decisive when isolated and susceptible to evaluation only with other clinical evidence the following may also be mentioned

1 Flutter and fibrillation
2 Paroxysmal tachycardias
3 Parasystole



FIG (a) Electrocardiogram is almost normal (b) There is right bundle branch block.

All of the three electrocardiographic alterations just enumerated may also appear in patients who either continually or over a long period do not exhibit any obvious evidence of a cardiac affection But such cases arouse suspicion and always demand a very careful examination and observation

A normal electrocardiogram never proves that the heart is healthy

The *prognosis* of cardiac disease should never be determined from the electrocardiogram alone

In the following discussion information made available by the preceding sections is amplified with the aid of additional illustrations

Fig 252a shows the electrocardiogram of a twenty two year-old male who as a child had suffered from rheumatic fever and chorea Since that time an insufficiency of the aortic valve with a heart of normal size had existed In 1933 examination furnished the electro

XXVI

DIFFERENTIAL DIAGNOSIS OF ABNORMAL PATTERNS AND OF ARRHYTHMIAS

At numerous places in this book it has been pointed out that not rarely the diagnosis of involvement of the myocardium can be made with certainty *from the electrocardiogram alone*. With the assistance of numerous examples it has been demonstrated that the participation of the heart muscle, so common in acute rheumatic fever, infectious diseases, tonsillitis and coronary diseases, at times can be recognized *only* by means of the electrocardiogram while the clinical examination is negative. Not rarely the electrocardiogram proves that the persistent palpitation after tonsillitis or on infectious disease must be attributed to an accompanying myocarditis, that a vague pressure in the region of the heart accompanied by marked meteorism is the result of coronary sclerosis or that a severe coronary involvement exists in a patient affected by syphilis years before, although the present complaints are trifling.

But it has also been emphasized that digitalis therapy may alter the electrocardiogram so profoundly that the picture associated with severe myocardial injury appears. Reference has also been made to the fact that in some normal people who are standing quietly (indeed, in rare cases even when sitting) electrocardiograms are regularly found which must be unequivocally designated as abnormal.

It has been repeatedly stressed that one can conclude from the electrocardiogram that a myocardial disease is present, but no information as to its nature is obtained. Indeed there is no electrocardiogram typical of any pathologic process. The history and the physical examination usually assist in the differential diagnosis. The electrocardiogram supplements the result of clinical examinations but never supplants them.

Abnormalities and signs indicating that pathologic foci of some type (recent or old, inflammatory, degenerative scars, etc.) are definitely present in the heart are the following:

1. Initial deflections which are wider than 0.11 second.
2. Marked notching and slurring of the waves of the initial deflection;
3. Abnormal RS-T segments (observe the physiologic displacements and the disturbing effects of polarization and rule out the action of digitalis).

RS-T is well as a deep inverted T wave The diagnosis of myocardial damage seems indisputable (posterior wall infarction)

The electrocardiogram of the third case was obtained from a sixty three year-old male who twice had survived an apoplexy with transient hemiplegia In Lead I the electrocardiogram is normal In Lead II the T wave is abnormally small and follows a slightly depressed RS-T segment The deep wave in Lead III is a widened Q wave For these reasons one may also suggest that the myocardium is abnormal (probably coronary sclerosis posterior wall infarction)

The fourth electrocardiogram was recorded in a forty six year old patient with malignant nephrosclerosis gallop rhythm as well

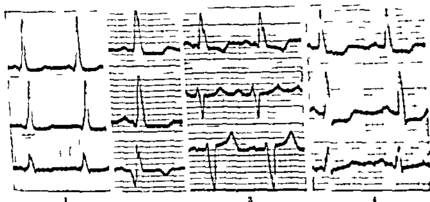


FIG 4 Abnormal electrocardiograms of four patients

as Cheyne Stokes breathing were present the non protein nitrogen was increased a short time after the electrocardiogram was taken the patient succumbed to his malady The electrocardiogram does not correspond to the usual picture which is obtained from cases of severe long standing hypertension In Leads I and II a plump initial deflection is seen (but not in Lead III) the T wave of Lead I is absent and the RS-T segment is depressed

The fifth series is from a sixty three year old patient whose heart was perfectly normal in size and shape the sounds were pure But upon walking he always suffered from typical radiating pains which immediately vanished upon taking nitroglycerine Here also a deep Q wave is present in Lead III In Lead II an unequivocal S wave is present Striking and certainly abnormal is the low T wave in Lead I

In the sixth series another electrocardiogram is reproduced

cardiogram of Fig 252a The initial deflections are 0 08 second in width, the T waves in Lead II are low but there are no *indisputable* signs of myocardial disease The notching of the initial deflection near the base line in Lead I may also appear normally

Without having been ill in the interim and still free from symptoms, the patient returned once again for examination after a lapse of two years The electrocardiogram recorded at that time is shown in Fig 252b The low notched P waves have remained unchanged In Lead I a deep and wide S wave follows after a thin R wave the same is seen in Lead II In Lead III a wide double pointed R wave appears after a short Q wave The width of the initial deflection has increased to 0 13 second The terminal deflections are normal

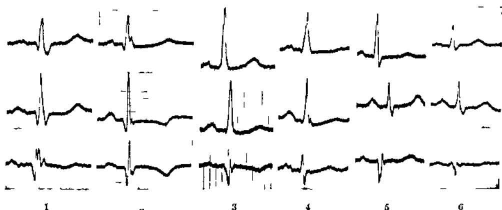


Fig 253 Electrocardiograms of six cases with signs of myocardial disease

The negative T wave in Lead III occurs normally The electrocardiogram shows the pattern of *right bundle branch block*

In Fig 253 the electrocardiograms of six patients are assembled (the three leads are shown beneath each other) all reveal signs of myocardial disease

At first glance Case 1 seems to show the typical picture of a right bundle branch block Nevertheless the presence of Q waves in *all* leads and the rather equal distribution of the widening of the initial deflection in Lead II suggest the possibility of some additional disturbance of intraventricular conduction The terminal deflections are normal in form At all events a disturbance of intraventricular conduction is present

In the second case the initial deflection is 0 10 second in width Notching near the base line is evident in all leads and the Q wave in Lead III is abnormally wide The terminal deflection in Lead I shows a slightly depressed RS-T in Lead II there is depressed

deflections are directed oppositely to the initial deflections One can assign electrocardiograms of this variety to the common form of bundle branch block But it should never be forgotten that another disturbance of intraventricular conduction without a block of the main stem may very easily produce the same kind of tracing A left axis deviation with widening of the initial deflections and abnormal oppositely directed terminal deflections is not rare in myocardial damage In this case a hypertension of 250/160 was present with a very large left heart gallop rhythm pulsus alternans and stasis

In the fourth electrocardiogram high P waves 0 09 second wide may be seen in each lead The RS-T segments are displaced below



Fig Case of coronary sclerosis (a) at rest (b) after exercise

the iso-electric line in all leads and the T waves are almost completely absent

The diagnosis of diffuse severe myocardial damage was confirmed by necropsy where an extensive myomalacia with coronary sclerosis was found

Fig 255 is that of a patient with typical attacks of angina pectoris The pain appeared after exertion of a trifling nature and compelled the patient to stand still The attacks were immediately abolished by amyl nitrite The electrocardiogram in 255a shows a left axis deviation the initial complexes are 0 10 second wide and somewhat plump The T waves are abnormally low in Lead I Fig 255b shows the electrocardiogram immediately after exercise (climbing stairs 20 metres in height) The T waves in Lead I have become deeply negative in Lead III they are considerably higher In Lead II an auricular extrasystole is visible Therefore the examination is unequivocally in favour of coronary stenosis Four months later coronary thrombosis occurred necropsy showed an occlusion of the ramus descendens anterior

which resembles a block of the right bundle branch (uncommon type) The findings which suggest it are the plump S wave in Leads I and II, the deep wave generally considered as a Q wave in Lead III (which often but not always is preceded by a small positive notch), the positive T waves in Leads I and II and the negative T wave in Lead III But, on the other hand, it must be stressed that the initial deflections are only 0 10 second in width and the plumpness and width of the S waves in Leads I and II as well as the R wave in Lead III, are only slightly evident

Such transitional forms are not rare and are interesting At all events a disturbance of intraventricular conduction is present in these cases

Sections of tracings of four cases are shown in Fig 254

The electrocardiogram of Case 1 is certainly abnormal The initial deflections are plump, but scarcely 0 10 second in width The RS–T segments are depressed below the iso electric line in all three leads and the T waves are very low In other words the record shows a kind of ventricular complex that is seen in myocardial damage However, a glance at the P waves and the auriculo ventricular conduction time reveals that the P waves precede the initial deflections by only a few hundredths of a second It concerns (owing to the presence of an abnormal P–R interval combined with abnormal ventricular complexes) a case of abnormal auriculo ventricular conduction (the bundle of Kent) that is a harmless anomaly For this reason it is not correct in this case to assume myocardial damage on the basis of abnormal ventricular complexes The patient suffered from attacks of paroxysmal tachycardia which are typical of this disturbance

The electrocardiogram of the second case in Fig 254 was re corded in a patient with severe coronary sclerosis and anginal symptoms Two months after registration of this electrocardio gram the patient died suddenly Plump initial deflections widened to 0 11 second are present, together with deep Q waves in Lead III and deeply depressed RS–T segments with very low T waves in Leads I and II The deep and wide Q wave and the negative T in Lead III permit diagnosis of a posterior wall infarction with certainty Negative T waves in Lead III may occur in healthy individuals The widening of the initial deflection the abnormal RS–T segments and the Q wave in Lead II rather prove an extensive injury of the heart

In the third case a left axis deviation exists The initial deflec tions are 0 10 second in width and are notched The terminal

exercise test A strain of the heart by climbing stairs did not produce electrocardiographic alterations but after a meal (which in itself places an increased demand upon the heart) there appeared severe alterations of the terminal deflections even at rest but especially after exertion

There are patients who feel anginal pain even after the first mouthfuls of food In other words it does not depend upon the amount of food nor upon a full stomach It is probable that vago vagal reflexes which influence the coronary arteries from the digestive tract (Dietrich and Schwiegk) participate in causing the poor blood supply to the heart The overload of the heart by the work of digestion must then be added



FIG Same patient as FIG 6 before (a) and after (b) exercise (full stomach)

Even in their first communications on the exercise test Goldhammer and Scherf made reference to the fact that occasionally climbing stairs alone need not produce electrocardiographic changes, whereas the same exertion after a meal may produce distinct alterations

A slightly accelerated sinus rhythm with a normal conduction time of 0 15 second is present in Fig 208a Lead I shows a deep Q wave followed by a high R in Leads II and III deep S waves exist The initial deflections are neither notched nor widened In Lead I the RS–T segment departs from the descending limb of the P wave *shortly before* the R wave has returned *to the* base line The T wave in Lead I is positive *In Lead II the* terminal deflection is normal in shape in Lead III the RS–T segment is depressed slightly under the iso-electric line

c a.

Fig 256a was recorded in a forty nine year old patient who noted at times especially after eating and sometimes also on rapid walking, a sense of pressure in the "pit of the stomach" The pain did not radiate was not invariably aggravated by movement and at times persisted for ten minutes The clinical examination yielded normal findings The electrocardiogram shows a conduction time perhaps at the upper limit of normal (most distinct in Lead II), the initial deflection in Lead I is somewhat plump, but otherwise the findings are normal After an exercise test one sees (256b) the initial deflection in Lead I somewhat smaller (lower position of the diaphragm) but the depression of RS–T in Lead II does not exceed the

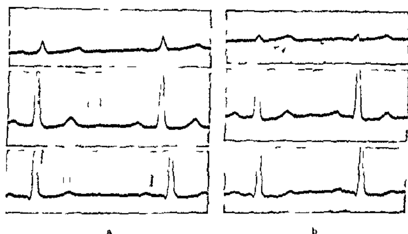


Fig 256 Electrocardiogram before and after work (stomach empty)

normal physiologic range, as often happens in the healthy the T wave in Lead I is even somewhat higher

On the basis of these results (which were obtained by others) the patient was treated as having gastritis and a corresponding diet and prescription were given He did not improve under the new therapeutic regimen and changed physicians

Since an electrocardiogram during rest and after work again presented the normal appearance (Fig 256a and b) another electrocardiogram (Fig 257a) was recorded after eating Distinct alterations became evident (*only* after eating and even before exercise) The RS–T segment in Lead II is slightly depressed below the iso electric line, and the T wave in Lead II is almost invisible An exercise test conducted immediately afterward showed a deep depression of the RS–T segment in Lead II (257b)

These tracings are instructive for several reasons At first they show how cautious one must be in the appraisal of the result of an

of the P wave so that it includes the conduction within the auricle

The other waves of the electrocardiogram in Fig 258c are normal

The electrocardiogram of Fig 259 was recorded in a twenty six year-old female who suffered from mitral stenosis Marked pulmonary stasis was present and severe attacks of pulmonary oedema appeared upon any excitement or physical exertion The right heart was not dilated and the left auricle was only slightly enlarged

The electrocardiogram shows Wenckebach periods In all leads may be noted an increasing prolongation of the auriculo ventricular intervals until a ventricular systole is dropped Corresponding to the slight alteration in cardiac size in this case, the

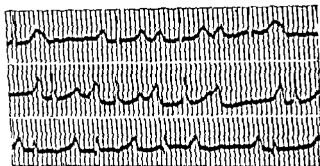


Fig 259 Electrocardiogram in patient with mitral stenosis and Wenckebach periods.

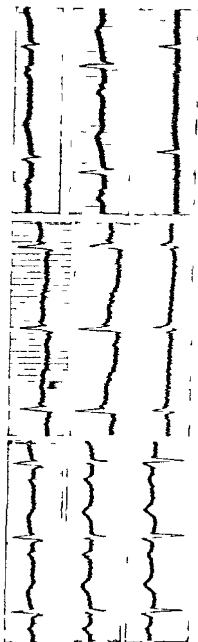
initial deflections are perfectly normal The unusually large P waves are also characteristic of mitral stenosis they attain an impressive height and width especially in Lead II The P–R segment so distinctly depressed below the iso-electric line indicates a very large after deflection of the P wave Study of the tracing also reveals that the depression of the RS–T segment which is found particularly in Lead II is also caused by the T of the P

In this case of mitral stenosis with marked pulmonary stasis the disturbance of conduction appeared after small doses of digitalis it was desirable since it led to a very useful diminution in the cardiac rate and consequently to a longer diastole

In Fig 260 a disturbance of rhythm is present In analysing the normal (sinus) beats it is well to begin after a long ventricular pause (before the third P wave in Lead I)

The P waves are normal in all leads Conduction time which is

The combination of a distinct Q wave, a slightly elevated take off of the terminal deflection in Lead I and the oppositely directed terminal deflection in Lead III is typical of an electrocardiogram of the Q_1T_1 type One may presume the presence of an antero lateral wall infarction (which in that case actually existed)



a b c

Fig 258 (a) Recent coronary thrombosis (b) Case of pericarditis (c) Simulated prolonged auriculo ventricular conduction actually caused by a disturbance of intra auricular conduction

It should not be forgotten that the presence of a Q wave without very definite alterations of the terminal deflection in the same lead supports the diagnosis of a coronary thrombosis In the absence of clinical signs the diagnosis of coronary thrombosis should not be made solely on the basis of the electrocardiogram since similar electrocardiograms also may appear in other conditions

In Fig 258b a high take off of the terminal deflection from the descending limb of the R again appears But in this tracing the alteration is present in *all* leads and is most marked in Lead II in coronary thrombosis it is found most distinctly either in Lead I *or* Lead III The electrocardiogram was recorded in a young man suffering from *lobar pneumonia with clinical* signs of pericarditis

In Fig 258c a prolongation of auriculo ventricular conduction time to 0 22 of a second is noted But more careful inspection reveals that the P waves are 0 12 second wide and this prolongs the conduction time *In other words the prolongation of conduction is simulated* simply by a disturbance of intra auricular conduction The conduction time is measured from the beginning

of the P wave so that it includes the conduction within the auricle

The other waves of the electrocardiogram in Fig 258c are normal

The electrocardiogram of Fig 259 was recorded in a twenty six year-old female who suffered from mitral stenosis Marked pulmonary stasis was present and severe attacks of pulmonary oedema appeared upon any excitement or physical exertion The right heart was not dilated and the left auricle was only slightly enlarged

The electrocardiogram shows Wenckebach periods In all leads may be noted an increasing prolongation of the auriculo ventricular intervals until a ventricular systole is dropped Corresponding to the slight alteration in cardiac size in this case the

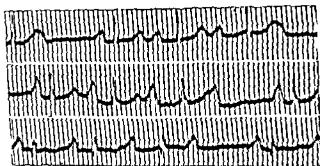


FIG 259 Electrocardiogram in patient with mitral stenosis and Wenckebach periods.

initial deflections are perfectly normal The unusually large P waves are also characteristic of mitral stenosis they attain an impressive height and width especially in Lead II The P-R segment so distinctly depressed below the iso-electric line indicates a very large after deflection of the P wave Study of the tracing also reveals that the depression of the RS-T segment which is found particularly in Lead II is also caused by the T of the P

In this case of mitral stenosis with marked pulmonary stasis the disturbance of conduction appeared after small doses of digitalis it was desirable since it led to a very useful diminution in the cardiac rate and consequently to a longer diastole

In Fig 260 a disturbance of rhythm is present In analysing the normal (sinus) beats it is well to begin after a long ventricular pause (before the third R wave in Lead I)

The P waves are normal in all leads Conduction time which is

best measured in Lead II amounts to 0 22 second The initial deflection in Lead III reveals a deep Q wave It is 0 09 second in width and somewhat plump The RS–T segment in Lead I is slightly depressed below the iso electric line and is followed by a very low T wave

In Lead II the T wave is absent, in Lead III a high take off of the terminal deflection from the descending limb of the R wave passes over into a negative T wave

The curve shows definite evidence of a myocardial affection, the patient reported that four days previously he had suffered from an extremely severe pain which lasted for several hours, it was felt at the lower end of the sternum and radiated upward into the neck The presence of a recent coronary thrombosis which



Fig 260 Signs of posterior wall infarction and ventricular extrasystoles

was situated in the descending ramus of the right coronary artery (posterior wall infarction) may be assumed

Moreover ventricular extrasystoles appeared after every normal beat at times after every two normal beats they showed no variation in form According to the rules given in the chapter on the localization of extrasystoles they probably originated in the right ventricle near the apex

The appearance of ventricular extrasystoles in a case of recent coronary occlusion is always an undesirable complication, patients affected in this manner must be watched carefully and treated with quinidine The transition of multiple ventricular extrasystoles into tachycardia is not rare and the danger of ventricular fibrillation must be considered

Fig 261 is the electrocardiogram of a patient who was under treatment for tabes in a neurologic clinic One day pain of excruciating severity suddenly developed in the cardiac region and per

sisted The patient came to the neurologic clinic, from which he was referred to the hospital The history stated that for some time pain had occurred after physical exertion

The electrocardiogram reveals auricular fibrillation with very rapid ventricular rate (170 to 180) A high take-off of the RS–T segment from the descending limb of the R wave may be seen in Lead I Alterations are also visible in an unusually clear manner in Leads II and III where there *is a low take-off* of the RS–T from the ascending limb of the S waves In conjunction with the history the electrocardiogram is in favour of coronary *thrombosis*

Without any relief or improvement from therapy, the patient died a few hours later at necropsy it was found that both coronary arteries were quite normal except at the orifices There was a syphi

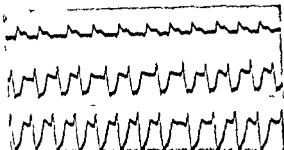


Fig 61 Auricular fibrillation and the electrocardiogram of coronary thrombosis in a patient with luetic coronary stenosis.

litic aortitis with such an extreme narrowing of the ostium of the left coronary artery that it was hardly permeable to a sound of hair like diameter Since the ostium of the right coronary artery was also markedly narrowed one must assume that even at rest only a very limited coronary perfusion was present With the appearance of paroxysmal auricular fibrillation and the associated rapid rate of the ventricle the disproportion between the oxygen need of the muscle and the oxygen supply was so great that clinically and electrocardiographically the picture of an acute coronary occlusion was simulated

A disturbance of rhythm is also present in Fig 262 The P waves of the normal beats (see Lead II) show varying forms (intra auricular conduction disturbance) The auriculo ventricular conduction time is normal The QRS complexes show a left axis deviation are 0 09 second in width and appear plump The terminal deflections are directed opposite to the QRS complexes the RS–T segments and

T waves accordingly are depressed below the iso electric line in Leads I and II, while they are above the base line in Lead III

Moreover, ventricular extrasystoles are present and in part are *multiple* Thus one notes that in Lead I three extrasystoles follow

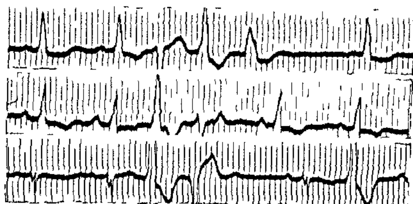


FIG 262 Signs of myocardial disease and ventricular extrasystoles of varying appearance

soon after the second normal beat The extrasystoles show varying *form* The patient was not receiving digitalis and a myocardial injury must be assumed on the basis of the appearance of the multiform extrasystoles

Fig 263 shows in the upper series normal sinus rhythm with normal conduction time The initial deflections are 0 11 of a second



FIG 263 (Upper) A disturbance of intraventricular conduction (Lower) A paroxysmal (auriculo ventricular ?) tachycardia

wide notched and plump The terminal deflections are normal in form Accordingly, a disturbance of intraventricular conduction is present In the lower series a regular tachycardia may be seen whose rate to be sure amounts to only 125 beats per minute P waves are not visible, the initial and terminal deflections show the same form in all details as the ventricular complexes during sinus rhythm In other words, a tachycardia is present arising from above the site of the bifurcation of the auriculo ventricular system (that

is in the A-V node or the bundle of His) The attacks lasted from some minutes up to an hour and usually appeared after physical exertion

One month before the electrocardiogram shown in Fig 264 was taken the patient had noted severe pain behind the sternum lasting for six hours In the following few days the typical clinical signs which are usually encountered in a case of coronary thrombosis were noted

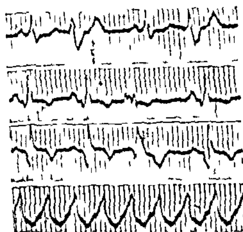


Fig 264 Electrocardiogram of coronary thrombosis with ventricular extrasystoles Lowermost tracing shows paroxysmal ventricular tachycardia.

In Fig 264 a sinus rhythm is interrupted by numerous extrasystoles The P waves are normal the conduction time amounts to 0 15 second The initial deflections of the sinus beats in Lead III show a very deep Q wave They are 0 10 second wide and plump In Lead I the RS-T segment is slightly depressed and the T wave is normal In Lead II and especially in Lead III a deeply inverted T wave may be seen Therefore the electrocardiogram is an instance of the Q_3T_3 type which is ordinarily seen in posterior wall infarction Moreover ventricular extrasystoles (base of the left ventricle) are present In the lowermost record a short section of a tracing from the same patient may be seen in Lead III it was recorded during an attack of paroxysmal ventricular tachycardia

Auricular fibrillation is present in Fig 265 The ventricular complexes of some stimuli conducted from the auricle to the ventricle show a left axis deviation and slightly depressed RS-T segments in Leads I and II The T waves are very low In addition abnormal ventricular complexes appear (the last three in Lead I the second in Lead II and the first in Lead III) In all respects these resemble the picture of the uncommon type of bundle branch block as shown in Figs 32 and 33 These beats came quite irregularly and accordingly were conducted beats and do not represent extrasystoles (intermittent bundle branch block)

In Fig 266 after the first and second normal beats two auricular

extrasystoles appear, and after the third normal beat four extrasystoles are noted The negative P wave of the extrasystole is seen distinctly at the end of the preceding T wave

The first extrasystole of a group is always conducted slowly (prolonged P–R interval) as well as aberrantly (abnormal ventricular complex) to the ventricle This is striking because the first ventricular complex of a group of extrasystoles does not appear more prematurely than the subsequent ones which are conducted normally Thus one sees that the stimulus of the first auricular extrasystole conducted abnormally in the ventricle, usually reaches the ventricle

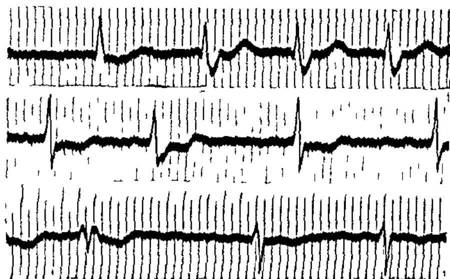


FIG 26 Auricular fibrillation and intermittent bundle branch block

after 0 45 second but on the contrary the second normally conducted auricular extrasystole reaches the ventricle even after 0 40 second

The first auricular extrasystole (the first negative P wave) may well emerge earlier in diastole than the subsequent extrasystoles However as measurement reveals the ventricle itself or the conduction system below the site of bifurcation of the bundle is not reached abnormally early by the first auricular extrasystole

The particularly poor conduction of the first extrasystole is only *one* example of a general law if several conductions follow each other after a long cardiac pause the second conducted beat finds an especially unfavourable condition of the auriculo ventricular and intraventricular conduction system This behaviour can rarely

be observed with a normal state of the heart But it is distinctly evident when the heart works under adverse conditions of disease or intoxication (digitalis)

The reason for this phenomenon seems to be the fact that the first systole after a long cardiac pause is especially powerful and is therefore accompanied by a longer refractory period If the following beat arrives very soon it finds very unfavourable conditions of conduction All subsequent beats which follow one another after a shorter cardiac pause are weaker and have a shorter refractory phase so that the conditions for conduction are much better even if many beats (for example in a tachycardia) appear This law explains why in a paroxysmal tachycardia the first extrasystole of the tachycardia so frequently exhibits a different form from the others and also accounts for the fact that usually in a Wenckebach period the second conduction is much more prolonged in comparison to the first one than is the third conduction compared to the second etc

The electrocardiogram of Fig 266 is certainly abnormal The RS-T segments in Leads I and II are depressed below the iso-electric line and the T waves in the same leads are very low The tracing is that of a fifty three year old woman who two years before had passed the menopause Severe flushes and palpitation were present as well as cardiac pain which tormented the patient exceedingly The blood pressure was 165/80

The abnormal electrocardiogram in combination with the pain in the cardiac region made the assumption of an organic cardiac disease (coronary sclerosis) seem likely But recent experiences have indicated that these electrocardiograms are frequently seen in women with ovarian insufficiency and that adequate treatment with estrogenic hormone not only abolishes the symptoms but also permits the electrocardiogram to return to normal These electrocardiograms are found in the natural and artificial menopause and in young women with hypogenitalism and disturbances of menstruation

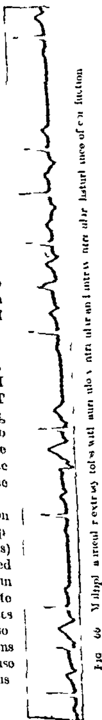


Fig 66 Multiple auricular extrasystoles with auriculo ventricular and intraventricular disturbance of conduction

It is *probable* that the condition is due to a disturbance of *nutrition of the* myocardium caused by a disturbance in endocrine balance and induced *prematurely* by the loss of ovarian hormone (*hyperfunction of the hypophysis*, thyroid, and adrenals) One must avoid confusion with the abnormal electrocardiograms *of myo* carditis, coronary sclerosis result of digitalis therapy etc which are similar in appearance

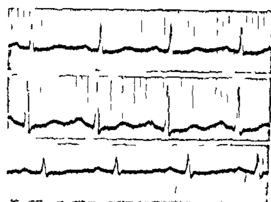


Fig 267 Abnormal electrocardiogram in climacterium

Fig 268 was obtained from a girl ten years old, who reported having been well until she had an attack of whooping cough one year prior to examination Since then attacks of palpitation had occurred they appeared suddenly and ended *just* as abruptly after lasting from minutes to hours Since more recent attacks had been more prolonged and manifes*tations of congestive failure* developed, medical aid was sought

Fig 268 *shows* the three leads during one of the attacks, which (*as may readily* be perceived from the large positive P waves) were caused by an auricular tachycardia with a rate of 206 beats per minute The auricular beats were followed by two kinds of ventricular contractions (1) initial deflections abnormal in shape wide and notched, which appeared after a conduction time of 0 08 to 0 10 second (2) ventricular complexes which were entirely normal in shape and appeared after a conduction time prolonged to 0 24 second But as should be stated immediately, this prolonga tion was the result of digitalis therapy and was never seen when digitalis had not been given The remaining waves were not modi fied by the employment of digitalis only the T wave became depressed below the iso electric line The two types of QRS com plexes were repeatedly recorded interchangeably in the intervals between attacks for minutes or hours during slow sinus rhythm a normal electrocardiogram or abnormal ventricular beats appeared the latter after a shortened conduction time All ventricular beats were produced by stimuli conducted from the auricles In other words an abnormal auriculo ventricular connection such as was described on p 418 (Kent s bundle) was present

In Fig 268 normally conducted beats are evident at the begin

ning of Lead I and at the end of Lead II In Lead III only normally conducted beats are present The disturbance in the second section of Lead III is an artefact Conduction between the auricle and the ventricle is disturbed After a series of normally conducted beats a 2 1 or 3 2 block recurs repeatedly

The tracing of Fig 268 is of interest for several reasons A paroxysmal tachycardia is present and during the tachycardia both forms of conduction appeared This finding is decisively against de Boer s theory of the origin of paroxysmal tachycardia by the circulation of an excitation wave between the auricle and the

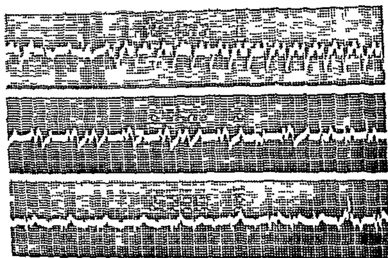


Fig 68 Bundle of Kent with Wenckebach periods

ventricle along the bundles of His and of Kent (p 423) Moreover in Fig 268 it may be noted (Lead II) that during the failure of conduction in one path the other pathway may be employed Of extreme interest is the appearance of a prolongation of the auriculo ventricular conduction *time in the* second conducted abnormal beat of the two 3 2 block groups in Lead II *In other words, a Wenckebach period can also appear in the abnormal path* The second stimulus requires approximately 0 05 second longer for conduction than does the first

We believe that this observation proves that the abnormal beats actually are conducted normal auricular stimuli this is against numerous other suggestions put forward to explain this disturbance

In the preceding discussion the term 'bundle of Kent' h been employed to designate electrocardiograms presenting certa

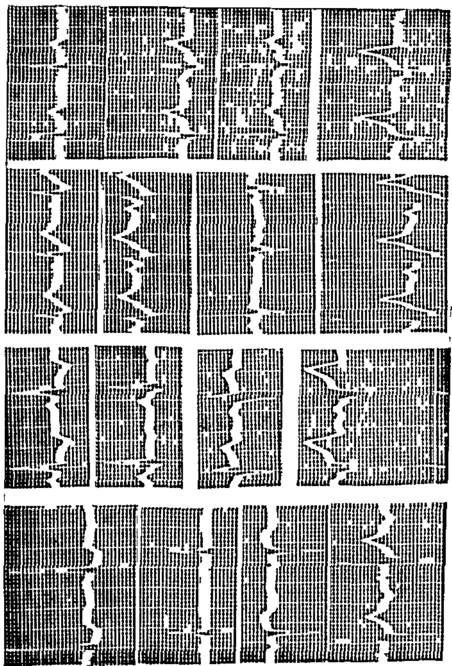


Fig 269 Leads from extremities and chest (CR_4) obtained from four patients (D

features However it should be emphasized that this is merely an expression of convenience there is no experimental or clinical

evidence that the right lateral bundle is actually or exclusively responsible for the disturbance It is quite possible that some other as yet unknown pathway is employed

Fig 269 shows the leads from the extremities *and chest* (CR_4) from four patients The first series was obtained from a seventy year old patient with hypertension He had received digitalis in the dose of 0 15 gm daily for three weeks The conventional leads show a left axis deviation with RS-T segments and T waves directed opposite to the main deflection The alteration of the RS-T segment is not dependent solely upon left ventricular strain digitalis is probably responsible in part The chest lead is normal In the leads from the extremities and especially in the chest leads *inverted* U waves are visible They are never found in the healthy individual

The second series show the electrocardiogram of a fifty six year old patient with hypertension No story of anginal pain was obtainable Leads I and II show the typical changes of a long standing hypertension with depression of the RS-T segment and inversion of the T waves The deep Q wave in Lead III however is an added complication Such electrocardiograms are common in old hypertensive patients with myocardial infarction There is no P wave in the chest lead (CR_2)

The third series was obtained from a fifty-one year-old patient with coronary sclerosis There is a bundle branch block with widening of the QRS complex to 0 13 second In Leads I and II the widening involves only the S wave This is often overlooked and the electrocardiogram is considered normal since the T waves are positive

The fourth tracing obtained from a seventy three year old patient shows a normal electrocardiogram in the leads from the extremities In the chest lead there is only one unusual feature namely the short Q wave preceding the R In tracings taken from the precordium at the apex however such a short Q wave may be found occasionally in the absence of a heart lesion

Fig 270 shows three tracings from different patients with acute rheumatic pericarditis The first record was secured from a thirty four year old man and shows a prolonged P-R interval and a slight elevation of the RS-T segment in Leads I and II While an elevation equal to this may occasionally be seen in the healthy subject it is never associated with such low voltage As a matter of fact if a physiologic elevation of the RS-T segment occurs the voltage of the QRS complex is much higher The clinical observation however and the alterations of subsequent electrocardiograms sug

gested an acute pericarditis The second tracing is that of a sixteen year old girl In Lead I it shows typical bifid T waves which are *so frequently observed in the late stages of a rheumatic pericarditis*

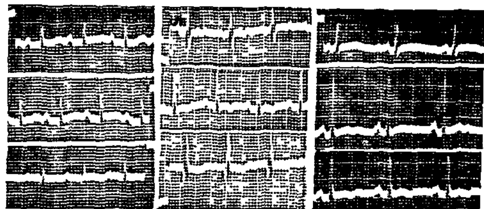


FIG 270 Tracings taken from three patients with rheumatic pericarditis

The third tracing was obtained from a thirty eight year old woman with rheumatic fever It shows changes similar to those in the second case The T wave in Lead I is abnormally low and it is bifid in Lead II Such T waves are readily and frequently confused with U waves

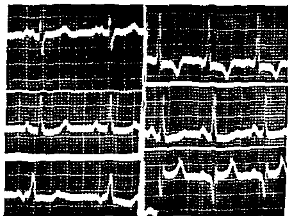


FIG 271 (a) From patient with mitral stenosis (b) From hypertensive patient with coronary sclerosis

The first series of Fig 271 was obtained from a patient with mitral stenosis *There is typical* widening and notching of the P waves and a right axis deviation The slight elevation of the RS-T segment in Lead I and depression in Lead III are due to right

ventricular strain The second tracing in this figure was obtained from a forty two year old patient with hypertension of the 'malignant' type Leads I and II show typical left ventricular strain, in Lead III however a deep Q wave appears and in this case proves the existence of a coronary sclerosis One is not permitted to diagnose a posterior wall infarction in this type of patient, these tracings are rather common

The upper tracing of Fig 272 shows a ventricular extrasystole (Lead III) Measurement of the post-extrasystolic pause reveals that it is not compensatory The distance *between the last beat* before and the first normal beat after the extrasystole is shorter



Fig 272 (Upper) Ventricular extrasystole reversely conducted to auricle (Lower) Ventricular tachycardia in a complete heart block

than two normal periods Between the initial complex and the T wave of the extrasystole one sees an inverted P wave Therefore one may assume that the ventricular extrasystole is reversely conducted to the auricle when it reaches the sinus node the regular formation of the stimulus is disturbed Such reversed conduction is rare in man

The lower tracing in Fig 272 (Lead II) was obtained from a forty nine year old man with coronary sclerosis and complete heart block It shows the end of a paroxysmal ventricular tachycardia Observations of this kind are common In the chapter on Stokes Adams syndrome it was *pointed out that* a great many of the attacks of Stokes Adams in patients with heart block are due to paroxysmal ventricular tachycardia or even short attacks of ventricular fibrillation

The electrocardiogram in Fig 273a was obtained from a seventy four year old woman with moderate hypertension It shows a left axis deviation with the RS–T segments slightly displaced opposite

to the main deflection During hospitalization the patient developed signs of pulmonary embolism (confirmed at necropsy) The tracing

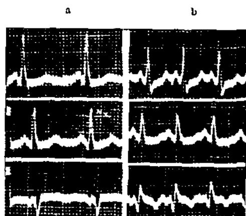


Fig 273 Before and after pulmonary embolism

recorded immediately after the appearance of acute symptoms (273b) shows changes typical for pulmonary embolism A deep

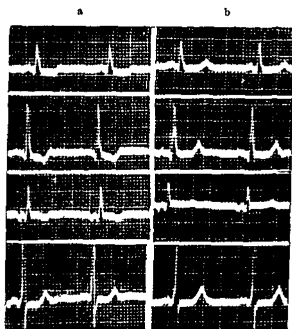


Fig 274 See text for description

S wave appeared in Lead I with a definite depression of the RS-T segment A Q wave, as well as inversion of the T wave, is present in Lead III

Fig 274a was obtained from a sixty seven year-old obese woman who had been under treatment for dyspnœa The electrocardiogram shows a regular sinus rhythm with a normal conduction time The RS-T segments are depressed below the base line in every lead Since the patient had received digitalis prior to admission the RS-T segment shows the slow descent and rapid ascent typical for this drug The Q-T interval is shortened Accordingly it was assumed that the alterations might be assigned to digitalis a tracing obtained fifteen days after digitalis administration had been stopped was completely normal (274b) There was no evidence of organic heart disease

XXVII

VECTORCARDIOGRAPHY AND VECTOR-ANALYSIS OF THE ELECTROCARDIOGRAM

The electrocardiographic Leads discussed in the preceding chapters are called scalar leads since they are derived from forces having magnitude and sense It has been known since Einthoven that the electric forces created by the activity of the heart may at any moment be represented by a line which has magnitude sense and position This is a *vector*

A series of data were given in the chapter on axis deviation and hypertrophy (p 87) Actually the so called electrical axis is a vector For the sake of clarity some of the facts mentioned in preceding sections of the book will be repeated in the following pages and considerable new data will be added in order to provide an introduction to the vector methods which are of value for under standing the electrical activity of the heart

VECTOR PRINCIPLES

It has been shown (p 87) that at any moment all electrical forces which reach the surface of the body (that is those which do not neutralize each other) may be represented by a vector which Einthoven called the Manifest resultant potential of the electrical cardiac activity This vector can be found by drawing perpendiculars from the projection of the leads on the sides of the Einthoven triangle (p 48) and reversely the form of the waves of the QRS complexes can be determined by projection from the vector It has been shown also that in addition to the mean vector (or axis) instantaneous axes (p 87) can be obtained from the height and direction of the waves at intervals of 0 01 second If the tips of the vectors are united one obtains a loop called the vectorcardiogram or monocardiogram as it was named by Mann the first to make this attempt With the introduction of the cathode ray oscillograph the vectorcardiogram could be registered directly (Schellong Wilson and Johnston) In recent years particularly due to the investigations of Duchosal and Sulzer great progress has been made in this field (Jouve Donzelot Milovanovich Grishman *et al*)

In order to use the method clinically it is necessary to make certain assumptions which are obviously inaccurate but serve their purpose It is assumed that the body constitutes a volume conductor

which is large in comparison to the heart and that the conductivity of the tissues is uniform (actually some tissues the lung for instance are apparently very poor conductors) It is assumed that the limb electrodes mark the apices of an equilateral triangle with the heart in its centre equidistant from the apices Finally, despite the fact that at any moment many forces are created in different parts of the heart for the sake of simplicity all the activity is assumed to be concentrated in and emanating from the centre of the heart

The vector when it stands as a mathematical symbol ($\vec{F}$) for the electrical forces created in the heart has its position in the

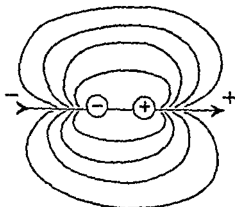


Fig 275 Diagram of relationship between the electrical field of force its origin (dipole) and its representation as a vector (arrow) its head is on the positive side its tail on the negative its length is proportional to the magnitude of the electrical forces.

centre of the heart and its direction is such that it points to the positive side its length is proportional to the magnitude of the electrical force It is often convenient to think of the vector as an arrow which represents a dipole the arrowhead being the positive pole and the tail of the arrow being the negative pole (Fig 275)

To find the resultant of several vectors (*R*) for instance of the vectors m and n acting together one can use the parallelogram method (Fig 276B) or one can simply place the arrows end on end (Fig 276C)

The physiological meaning of the *heart* vector depends on the phase of activity which it represents During the activation of the auricle (P wave) the vector represents the manifest (not neutralized) potentials of the auricles and indicates the direction in which this activation is proceeding A vector obtained during the activation

of the ventricles (QRS complex) shows the direction of their activation During the recovery phase (RS–T and T) the vector *points* opposite to the direction in which recovery is proceeding During the activation phase it points to the myocardium not yet activated, during the recovery phase it points to the myocardium which has already recovered (It was noted on p 2 that the positive pole

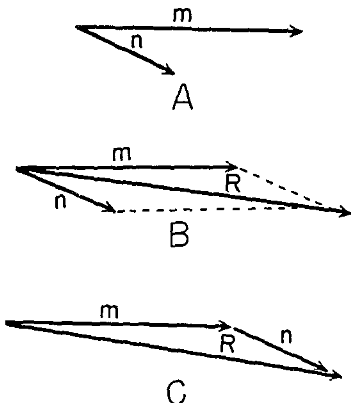


Fig 276 Two graphic methods of adding vectors m and n are the two vectors to be added R is the resultant vector Fig 276B shows the parallelogram method Fig 276C shows the end to end method

is in front of the activation wave while the negative pole is in front of the recovery or repolarization wave) In each instance these directions of the vector are due to the fact that the resting muscle is positively charged as compared to the activated depolarized muscle

The term heart vector will be used to denote the vector of any electrical force originating in the heart An instantaneous vector is one which is present at any given instant A mean vector represents the average of all vectors present during a given period for instance during the activation time of the ventricles The term

integral vector usually is synonymous with instantaneous but carries the implication that it is the resultant of all the innumerable small vectors which are simultaneously active in different parts of the heart All vectors are usually placed in the centre of the heart and accordingly also in the centre of the triangle

The heart vector is the source of the electrical field which permeates the body with its lines of force (Fig 277) An electrode

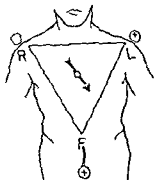


Fig 7 The charge which an electrode picks up from a given electrical force can be predicted by studying the relationship of the electrode to the vector which represents the force The Einthoven equilateral triangle is superimposed on the trunk the apices stand for the three limb electrodes right arm (R) left arm (L) and left leg (F) O is the centre of the heart of the triangle of the body and the vector R is close to the tail than to the head therefore its charge is negative The net charge of L is zero since F is close to the head its charge is positive

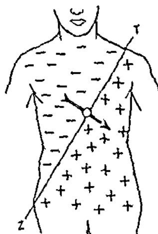


Fig 8 *Diagram* showing how the *transitional* plane divides the body into positive and negative parts for a given heart vector An electrode anywhere to one side of it will receive negative charges *on* the other side of the transitional plane it will pick up positive *charges* An electrode on the transitional plane will have a net charge of zero

which is closer to the positive tip of the vector will pick up a positive charge (Fig 277) one closer to the negative tip a negative charge If the electrode is equidistant from the *tips of* the heart vector it will be at zero potential or its equivalent that is a combination of positive and negative charges which add up to zero Accordingly an electrode anywhere *on the plane* which perpendicularly bisects the heart vector will be at zero potential (Fig 278) This plane is the zero plane or the transitional plane It divides the body into positive and negative parts Where the plane intersects the body

of the ventricles (QRS complex) shows the direction of their activation During the recovery phase (RS–T and T) the vector points opposite to the direction in which recovery is proceeding During the activation phase it points to the myocardium not yet activated during the recovery phase it points to the myocardium which has already recovered (It was noted on p 2 that the positive pole

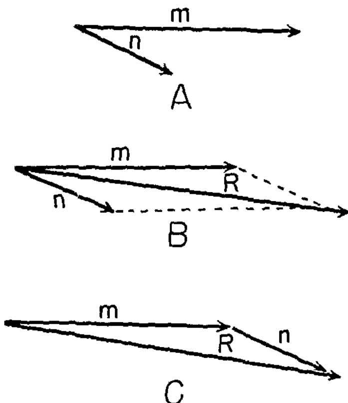


Fig 276 Two graphic methods of adding vectors m and n are the two vectors to be added R is the resultant vector Fig 276B shows the parallelogram method Fig 276C shows the end to end method

is in front of the activation wave, while the negative pole is in front of the recovery or repolarization wave) In each instance these directions of the vector are due to the fact that the resting muscle is positively charged as compared to the activated depolarized muscle

The term heart vector will be used to denote the vector of any electrical force originating in the heart An instantaneous vector is one which is present at any given instant A mean vector represents the average of all vectors present during a given period for instance during the activation time of the ventricles The term

midline (Fig. 280c). The axis of V_2 is approximately sagittal, that of V_6 is approximately horizontal.

The axis of an *augmented* unipolar limb lead going through the centre of the triangle is prolonged until it bisects the opposite side

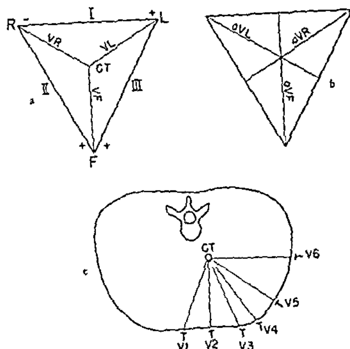


Fig. 280. The axes of the standard bipolar limb leads are shown as the sides of the equilateral triangle. The axes of the unipolar limb leads are shown as lines from the designated limb to the centre of the triangle (Fig. 280a). In Fig. 280b the axes of the augmented unipolar limb leads are shown as lines from the designated limb to the midpoint of the opposite side. In Fig. 280c the axes of the unipolar chest leads are shown as lines from the exploring electrode to the centre of the heart (i.e. to the centre of the cross section). CT designates the central terminal of Wilson. Its anatomical location for the purpose of vector projection is the centre of the heart. The latter lies in the centre of the equilateral triangle and somewhat to the left of the midline in the cross section of the thorax. Note that V_2 is shown as a true sagittal lead; this is, however, not true for all cases. Note also that V_6 and Lead I are both horizontal.

of the triangle (Fig. 280b). This indicates that the central terminal in these leads is the average of the two other unipolar limb leads. For the Einthoven triangle it is merely an elongation of the axis of the unipolar limb lead. This illustrates another principle: the size of the deflection is proportional to the length of the axis of the lead

wall it creates the transitional zone An exploring electrode on the transitional zone is equidistant from the tips of the vector and will record a zero or transitional deflection An electrode on the positive side of the zone will pick up a positive charge, one on the negative side a negative charge

There is another way to express the effect of a vector on an electrode when the vector points to an electrode it induces a positive charge and when it points away from the electrode a negative charge is induced, when it points neither to nor away the induced charge is zero or its equivalent, as defined above (Fig 279) One may therefore say (as it has been said earlier in this book) that when an activation front approaches an electrode it becomes positively charged when a recovery front approaches an electrode it becomes negatively charged

Fig 279 When the vector points to an electrode it imparts a positive charge when it points away a negative charge When it neither approaches toward nor recedes from the electrode the latter will be at zero potential

In order to correlate the deflections registered by electrocardiographic lead with the heart vector one projects the vector on the *axis of the lead* (p 47) As Fig 280 illustrates the axis of a standard lead is the line joining its two electrodes the heart being in the centre of the triangle Therefore the sides of the Einthoven triangle represent geometrically the axes of the three standard bipolar leads These are "poled" in a uniform manner as pointed out before so that when the deflection is positive (upright) in Lead I L is more positive than R when the deflection is positive in Lead II F is more positive than R finally when the deflection is positive in Lead III F is more positive than L Thus in Lead I the positive electrode is L and in Leads II and III the positive electrode is F

The axis of a "unipolar" lead is a line drawn from the exploring electrode to the centre of the heart The latter can be considered the location of the central terminal because both are at zero potential Even though the heart vector constantly changes direction during the cardiac cycle its pivot in the centre of the heart always remains at zero potential

The axis of a unipolar limb lead is a line from the designated apex of the triangle to the centre (Fig 280a) For the unipolar chest lead the lead line is drawn from the position of the exploring electrode to the centre of the heart which lies slightly to the left of

All vectors that lie on one side of the line will project positively on the given lead and all vectors on the other side will project negatively. Conversely if the deflection in a given lead is positive then the responsible heart vector must lie on the positive side of the line and *vice versa*.

In this manner one can correlate the heart vector with the deflection in a given lead easily and without anything more elaborate than a free hand sketch of the triangle a cross section or sagittal section of the thorax (see later). Mental geometry will usually suffice once this method is understood. For example if Lead I

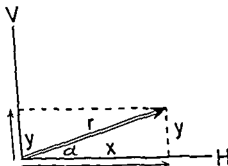


Fig 8 Trigonometry of projection. The cosine of angle alpha is x/r, sine of angle alpha is y/r. If r is the vector and its length is equal to 1 then the projection of the vector on the horizontal axis is equal to x and on the vertical axis equal to y. Accordingly when ever one finds the projection of cosine of an angle it indicates the projection of the vector on the x axis and sine of the angle indicates the projection on the y axis. *In each case the angle alpha indicates the angle* between the vector and the x axis

shows a positive wave then the vector must point to the left. It may point up or down forward or backward but always to the left. If Lead aVF shows a positive deflection the vector must point down. If both Lead I and Lead aVF show a positive deflection the vector will therefore be localized into the left lower quadrant of the frontal plane. The more leads used the narrower the sector in which the vector can be placed.

If two leads have parallel axes the deflection induced by a given vector will have the same polarity in both for their projection has the same polarity. However the magnitude will vary inversely with the distance of the vector from the lead axes and directly with the length of the lead axes.

Leads which may have parallel axes are Leads I Lead A (cube system see later) and V_6 (see Fig 280c). Leads V_2 V_8 and B (cube

Comparison of Fig 280a and Fig 280b shows that the augmented unipolar (aV) limb lead will register a deflection which is 1 5 times the size of a unipolar (V) limb lead

To project a vector on the axis of a lead, perpendiculars are dropped from the tips of the vector onto the axis of the lead In this way one creates a projected image of the vector on the axis of the lead, the manifest vector (see Fig 281) When the projected image points to the exploring electrode of a unipolar lead or to the positive electrode of a bipolar lead then the deflection registered by that lead will be positive and the vector is said to project positively on the lead (Fig 281) When it points away from the

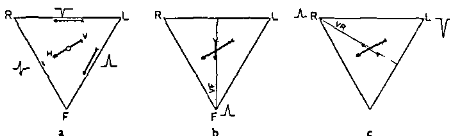


Fig 281 Geometry of vector projection In the centre the heart vector which is to be projected Fig 281a The projected images of the vector on the axes of Leads I II and III indicate the deflection which these leads will register Fig 281b Projection on axis of aVF The projected image points to the exploring electrode the deflection is therefore positive Fig 281c Projection on aVR again the deflection is positive because the projected image points to the exploring electrode In Lead aVL whose axis is not drawn the vector is parallel to it and projects with its full length on it consequently the deflection is at a maximum and is negative

exploring or positive electrode respectively the deflection is negative and the vector is said to project negatively on the lead When the heart vector is perpendicular to the axis of the lead its projected image is a point and the deflection is equal to zero or is a group of deflections which contains as much positivity as negativity (Fig 281a)

When the heart vector is parallel to the axis of a lead the projected image attains its greatest size In general the size of a deflection in a given lead can be predicted by noting the tendency of the heart vector to be perpendicular or parallel to its axis

The size of the projected image (e) equals the size of the heart vector ($\hat{E}$) times the cosine of the angle (alpha) which the heart vector forms with the axis of the lead $e = \hat{E} \times \cos \text{alpha}$ (Fig 282)

If one draws from the centre of the heart a line that is perpendicular to the axis of a given lead, this line can serve as a boundary

The origin of each loop is in the centre spot and thus corresponds to the isoelectric line of the electrocardiogram

There is no unanimity as to the best method for registering the horizontal vertical and sagittal components of the heart vector

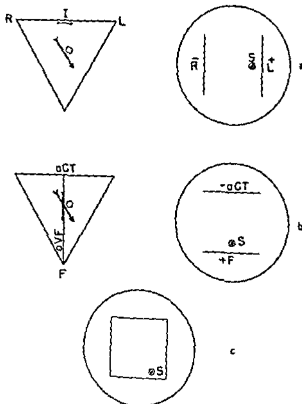


Fig 283 The light of the cathode ray oscilloscope indicates the position tip of the heart vector The electrode plates are connected (through an amplifying system) to the plates which cause the beam to deflect The projection of the heart vector on the lead indicates the influence of the vector on S the beam of electrons According to the leads used for the horizontal and vertical components one can obtain the projection of the spatial vector on the different planes

At present two systems of leads are popular the tetrahedral system (Wilson) and the cube system (Duchosal and Sulzer) (Fig 284)

In the tetrahedral system (Fig 284a) Lead I is the horizontal lead Lead VF or aVF the vertical and Lead V_2 the sagittal V_2 lies 3 cm to the left of the spine directly behind the centre of the

system) Leads aVF, VF and C (cube system) Actually these leads do not show identical deflections though they are very similar in most instances , there is often a close similarity in Leads I and V_6 The discrepancies can be explained in simple geometrical manner without recourse to complicated mathematical formulas

PRINCIPLES OF VECTORCARDIOGRAPHY

The vectorcardiogram is a record of the heart s electrical activity in the form of a tracing which connects the positive tips of a continuous series of instantaneous vectors It may either be constructed from the electrocardiogram or registered directly by means of a cathode ray oscilloscope In the latter a heated cathode emits a beam of electrons which impinges on a fluorescent screen and creates a spot of light Surrounding the beam are two pairs of electrically charged plates at right angles to each other as shown in Fig 283

If registration of the frontal projection of the vector is desired the upper and the lower plates are connected to the central terminal and Lead 1 respectively through an amplifying circuit to obtain the vertical component for the horizontal component R is con nected with the plate on the right and L with the plate on the left Thus the VI Lead whose axis is vertical is connected to the plates controlling the vertical motion of the beam (Fig 283b) and Lead I whose axis is horizontal is connected to the pair of electrodes controlling the horizontal motion (Fig 283a) According to the projection of the spatial vector on the two leads each of the plates acquires a charge The beam will be deviated to the plates with the positive charges In this manner at any given moment the spot of light seen on the screen represents the positive tip of the vector (Fig 283c) As the latter changes throughout the heart cycle the light inscribes a line usually in the form of a loop For each heart beat there are usually three loops corresponding to the P QRS and RS–T T phases of the cycle

The basic vector loop has however three dimensions and is called the spatial loop (sÊ) Only two dimensions are involved in the inscription of a loop on the screen this is equivalent to the projection of the spatial loop on the plane The vertical and horizontal leads as shown above form the frontal loop the horizontal and sagittal leads the horizontal loop (see later) and the sagittal and vertical leads the sagittal loop With the aid of these loops the spatial loop can be constructed

The origin of each loop is in the centre spot and this corresponds to the isoelectric line of the electrocardiogram

There is no unanimity as to the best method for registering the horizontal vertical and sagittal components of the heart vector

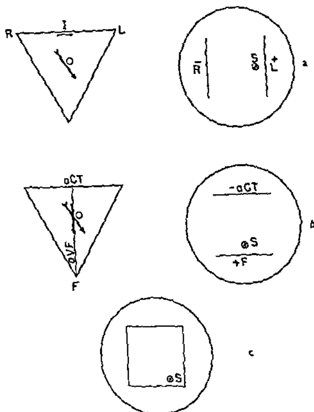


Fig 283 The light of the cathode ray oscilloscope indicates the positive tip of the heart vector The electrode plates are connected (through an amplifying system) to the plates which cause the beam to deflect The projection of the heart vector on the lead indicates the influence of the vector on the beam of electrons According to the leads used for the horizontal and vertical components one can obtain the projection of the spatial vector on the different planes.

At present two systems *of leads* are popular the tetrahedral system (Wilson) and the cube system (Duchosal and Sulzer) (Fig 284)

In the tetrahedral system (Fig 284a) Lead I is the horizontal lead Lead VF or aVF the vertical and Lead V_8 the sagittal V_8 lies 3 cm to the left of the spine directly behind the centre of the

ventricular mass If Lead I is registered at normal (N) sensitivity, then VF must be registered at 1 7 N, aVF at 1 15 N and V_8 at 1 2 N These sensitivity factors correct for the difference in the length of the axes of the different leads and for the difference in the distance of the heart vector from the leads

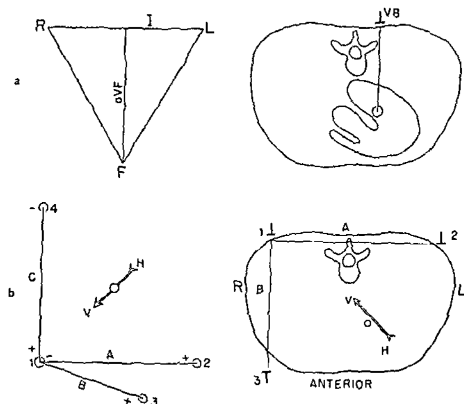


Fig 284 Fig 284a shows the tetrahedral system The horizontal lead is Lead I the vertical lead is Lead aVF and the sagittal is Lead V_8 Fig 284b indicates the trihedral or cube system Electrodes 1 2 and 3 are at the level of the 1st or 2nd lumbar vertebra 1 is in the right posterior axillary line 2 in the left posterior axillary line and 3 in the right anterior axillary line Electrode 4 is directly above 1 in the right suprascapular region The centre of the heart vector (o) is equidistant theoretically from each of the 4 electrodes The axes of the leads A (horizontal) B (sagittal) and C (vertical) are at right angles to each other

In the cube or trihedral system an electrode is placed on four points on the trunk so that they theoretically constitute the corners of a cube in whose centre lies the heart equidistant from each electrode and whose lead axes are equal in length (Fig 284b) Electrodes 1 2 and 3 lie at the level of the first or second lumbar vertebra Electrode 1 is in the right posterior axillary line 2 in

the left posterior axillary line and 3 in the left anterior axillary line Electrode 4 lies directly above 1 in the right suprascapular region

Lead A is the horizontal lead and equals 2 minus 1

Lead B is the sagittal lead and equals 3 minus 1

Lead C is the vertical lead and equals 1 minus 4

The tetrahedral method has the advantage of using standard electrode placements, while the cube method requires the use of four unorthodox and inconveniently located electrodes On the

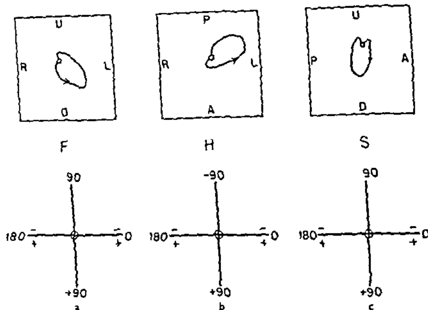


Fig 80 The orientation of the recording and the scheme of angle nomenclature which will be used F the frontal plane H the horizontal plane S the sagittal plane U means up D means down R indicates right L indicates left A anterior P posterior

other hand *several observers* have reported a better correlation between the horizontal loop and unipolar chest leads for the cube system than for the tetrahedral system This is a very important consideration in evaluating the accuracy of the lead system Of course the correlation between the frontal loop and limb leads will be perfect *for the loop recorded by the tetrahedral system* since the leads used in the construction of the loop are the limb leads

The eccentricity factor is less pronounced in the cube system than in the tetrahedral one However when the heart is very large in relation to the thorax as in the newborn and in adults with very

large hearts, this may not be true The definitive lead system has not yet been determined

Usually the loops registered by one method look very similar to those of the other The description of the normal and abnormal loop which follows will apply in general to loops recorded with either method The orientation of the loops in the frontal horizontal and sagittal plane is shown in Fig 285 Occasionally marked differences

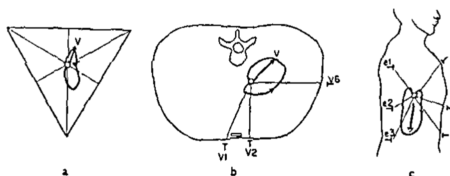


Fig 286 Correlation of the loop with the electrocardiogram Fig 286a the frontal loop is placed in the equilateral triangle O the origin is in the centre of the triangle Fig 286b the horizontal loop is placed in the cross section of the thorax with O to the left of the midline The *axis of each chest lead* is the line from the position of the exploring electrode to O Fig 286c the sagittal loop is placed in a diagram of the sagittal section of the trunk The method of drawing the axes of an œsophageal lead (e leads) is the same as for any other chest lead a line from the site of the exploring electrode to O the centre of the heart *In all three* diagrams one projects the instantaneous vector which is a line from O to a point of the loop on to the axis of any given lead The corresponding deflection in the electrocardiogram should agree in respect to polarization with the projection Thus in a the indicated heart vector OV projects positively on Leads I aVL and aVR it projects negatively on Leads II III and aVF In b another heart vector OV projects positively on V_6 and negatively on V_1 and V_2 on V it would have a zero projection For the instant designated by OV in b Leads V_1 and V_2 would show a negative V_4 an iso electric and V_6 a positive deflection In c the vector OV would project positively on œsophageal leads e2 and e3 and negatively on e1 Actually such corroboration usually is very satisfactory

are seen when different systems are used An analysis of the differences and their significance is still a subject for research

In order to correlate the spatial loop with the electrocardiogram the plane loop is placed in the appropriate reference system (Fig 286a) Thus the frontal loop is placed in the Einthoven equilateral triangle so that the origin lies in the centre of the triangle The horizontal loop is placed in a cross section of the thorax so that the origin of the loop is slightly to the left of the midline (Fig 286b) The sagittal loop is placed in a sagittal section of the thorax (Fig 286c) The axes of the different leads are indicated in the

diagrams A line from the origin of the loop to any point thereon represents the instaneous vector (its positive half only for the sake of simplicity) which is responsible for this point By projecting this vector on the axis of a lead one can ascertain the deflection which that vector should induce on that lead according to whether the vector is projected positively or negatively A comparison with the actual deflection recorded permits the correlation between the electrocardiogram and vectorcardiogram The frontal loop is correlated with the limb leads the horizontal loop with the chest leads and the sagittal loop with œsophageal leads and leads up and down the sternum and spine

The time factor is represented by interrupting the beam at regular intervals An electric tuning fork vibrating 400 times per second will cause the beam to be interrupted every 0 0025 second When the loop is inscribed rapidly the interruptions are widely spaced when it is inscribed slowly the interruptions are bunched together

It is important to emphasize that the loop is not a map of the conduction pathway Consequently when the loop is inscribed slowly it is not an expression of slow conduction It is true that an abnormally slow inscription of the loop often is the result of abnormal conduction and the latter is usually slower than normal however exceptions are frequent and one must not use the rate of inscription of the loop as an indication for the velocity of the conduction of the activation process

The direction in which the loop is written is described as clockwise or counterclockwise The direction is important It can be ascertained in several ways It may be seen directly on the screen however the movement often is so fast and complicated that this method is not very reliable Another method which appears excellent is to modulate the dashes so that their leading tip is narrower than the rear A third and very practical way is to open the loop by sweeping it across the screen as in Fig 287 A fourth method is to correlate the loop with the component electrocardiogram for example the frontal loop is compared with the horizontal and vertical leads This often permits a reliable determination of the direction of inscription but fails when the loop is complicated

The position of the loop is designated by the octant of space which it occupies (the same as pointed out for the electrical axes (Fig 42) The octants are described in terms of superior inferior right left and anterior posterior One can also describe the angle which the loop makes with the axes of a specific co ordinate system

Fig 285 shows the orientation of the loop and the co ordinate system which is used here

The loops projected on the screen are usually photographed, if possible the projections on the three planes are photographed so that the basic spatial loop can be reconstructed The loops may also be copied on paper during visualization on the screen For clinical purposes this is adequate, but must be done by the physician himself It is easy and instructive to construct the spatial loop with wire



Fig 287 By sweeping the loop across the screen the direction of inscription becomes clearer

The centre spot which contains the isoelectric point also embraces the greater portion of the P and RS–T, T loops and the very beginning and end of the QRS loop The result is a blob of light whose components can not be distinguished Magnification of the loops by increasing the sensitivity of the recording often resolves this mass into its components in a clear fashion

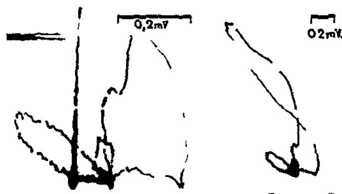


Fig 288 (From Duchosal and Sulzer) shows at the right a vectorcardiogram with normal sensitivity (1 mV 6 cm) and at the left the same vectorcardiogram with increased sensitivity (1 mV 20 cm) the P loops and T loops are clearly visible with magnification but only a part of the QRS loop could be obtained The vectorcardiogram points into an abnormal direction since the poling used is different than that used here

Fig 288 reproduced from Duchosal and Sulzer shows the loops registered with different amplifications With greater sensitivity the P and T loops are clearly visible Then as a rule only the beginning and end of the QRS loop is registered

Fig 289 shows the frontal vectorcardiogram as well as the reconstructed and the actually registered standard and unipolar limb leads. Note the similarity between the reconstructed and the registered leads. This figure is also reproduced from Duchosal and Sulzer.

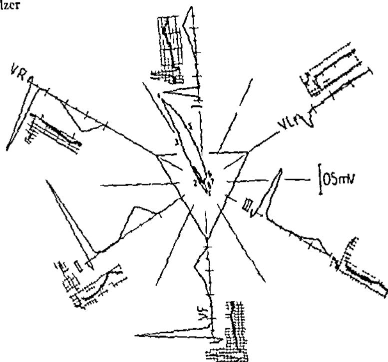


Fig. 289 (From Duchosal and Sulzer) Frontal vectorcardiogram pointing upward to the right because of the different poling used by the authors. The standard limb leads and the unipolar leads were constructed from this vectorcardiogram. There is a close correlation between these constructions and the electrocardiograms actually registered.

The physical meaning of the loop is obvious: it indicates the position of the positive tip of a series of instantaneous vectors. Its physiological significance, however, is not that simple. The loop is not a map of the pathway of activation or recovery. Only at the very beginning and end of a loop does the direction of the indicated instantaneous vector show the direction of the activation in a direct manner. For only at this time can one assume that a single area of the heart is active. At all other times the loop indicates the resultant of many component vectors arising simultaneously in various parts of the heart. It has been estimated that for the QRS

phase, these simultaneous vectors point in so many different directions and are so often opposed to each other that from 9/10 *to 24/25 of them* neutralize each other (Schaefer)

Should therefore one region of the myocardium fail to be activated then it will fail to contribute to the resultant vector the latter *will* point away from the missing area Accordingly in myocardial infarction the loop is deviated from the region which is *infarcted*

If a given region were activated in such a way that its small component vectors are parallel to each other (this can be called unanimous activation) then the resultant from this local region (papillary muscle for instance) will be so strong that it dominates the resultant from the rest of the heart Thus, if only 1/10 of the heart is activated in a unanimous fashion then the entire activity of the other 9/10 can be neutralized Accordingly the degree of an abnormality in the electrocardiogram is often not proportional to the size of the myocardium involved

Normally the loop is inscribed rapidly indicating that the instantaneous vector is changing rapidly in direction and size Should the vector remain fixed or the rate of change be slowed then the inscription of the loop will likewise be slowed The time interruptions would be crowded together in the recordings This may conceivably occur if a single region alone is activated in a normal manner but by itself Such an event may explain the occasional appearance of a terminal slow segment of the QRS loop which lies posteriorly to the right and superiorly (Schaffer)

CLINICAL VECTORCARDIOGRAPHY

The normal vectorcardiogram consists of a P QRS and the RS–T T loops Each loop usually is closed because normally each of these complexes begins and ends at the isoelectric line The ventricular loops are not closed in those cases where the RS–T segment is not in the zero line

The spatial P loop represents auricular activation The recovery phase of the auricles corresponding to the Ta wave usually is small and obscured by the QRS loop The P loop usually is small and difficult to study because it is buried in the centre spot When the loop is inscribed with higher magnification it lies to the left down and somewhat anteriorly It may be quite irregular Normal standards as to direction location form size regularity etc are not yet available so that at present their interpretation is based only on electrocardiographic principles

The QRS spatial loop normally appears somewhat elongated and es to the left and inferiorly In young subjects it tends to lie nteriorly in older ones more posteriorly It tends to be located n a single plane which usually is more sagittal than frontal or horizontal Its form is regular and smooth and the inscription is rapid and even A slow inscription which is revealed by the crowding together of the time markings may occasionally occur at the beginning or end of the loop An initial short segment usually proceeds anteriorly to the right and often superiorly The main centrifugal limb proceeds to the left and turns posteriorly to become the centripetal limb Often a small terminal segment which is slowly inscribed may appear posteriorly to the right and superiorly

The initial segment is ascribed to the activation of the septum from the left ventricular side to the right The centrifugal and centripetal limbs represent the changing instantaneous vectors as the body of the ventricles is activated from the inner to the outer layers and from the apical to the basal regions The terminal section may be due to the activation of a solitary area of the myocardium possibly in the base of the left ventricle No element of unusual delay is needed to explain this terminal portion Its slow inscription is easily explained by the absence of interference from other vectors since the entire ventricle has already been activated with the exception of this single region

The normal QRS loop as seen in the frontal plane (Fig 286) lies between 0 and 90 degrees and is usually inscribed clockwise There is a tendency for counterclockwise inscription when the loop is located farther to the left

Normally the horizontal QRS loop is always inscribed counterclockwise The initial segment to the right and anteriorly is readily noted (Fig 290) The centrifugal and centripetal limbs lie near the 0 axis to the left In the younger age groups much of the loop is anterior and in the older groups more is posterior The distinction between normal left axis deviation and mild left ventricular hypertrophy is not facilitated by inspection of the loop

The normal sagittal QRS loop is always inscribed clockwise For the most part it lies inferiorly the initial and terminal portions may be located superiorly but they are not large The location of the main portion of the loop anteriorly and posteriorly as noted previously tends to vary with the age Because the plane of the spatial QRS loop is for the most part sagittal the sagittal loop is usually as large or larger than frontal and horizontal loops

The normal RS–T T spatial loop tends to parallel the QRS loop

phase, these simultaneous vectors point in so many different directions and are so often opposed to each other that from 9/10 to 24/25 of them neutralize each other (Schaefer)

Should therefore one region of the *myocardium fail to be* activated then it will fail to contribute to the resultant vector the latter will point *away from* the missing area Accordingly in myocardial infarction the loop is deviated from the region which is infarcted

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CLINICAL VECTORCARDIOGRAPHY

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more slowly than the centripetal This corresponds to the flatter slope of the first part of the RS-T T complex and the steeper descent of the T wave to the zero line

It is almost impossible to discuss the RS-T T loop intelligently without reference to the concept of the gradient (see p 235) This requires as we saw estimation of the mean vector of a complex in terms of spatial direction magnitude (in millivolts) and duration (in seconds) From the vectorcardiogram the spatial direction and magnitude of the mean vector of a complex is directly visualized However the estimation of the duration of the complex is poorly represented for this one needs the electrocardiogram Therefore to evaluate the gradient completely one needs both types of records It appears that the vectorcardiogram will be very helpful in analysing the variations of the gradient resulting from various influences

The spatial QRS loop in *left ventricular hypertrophy* is located more to the left posteriorly and often more superiorly than normal (Fig 290) The spatial loop is unusually large and is inscribed rapidly without delay The initial segment is directed to the right and anteriorly like a normal loop The frontal loop is often inscribed clockwise and is located decidedly to the left and often superiorly The horizontal loop is always inscribed counterclockwise it is directed to the left and especially posteriorly The sagittal loop is inscribed clockwise and is located posteriorly and often superiorly The ST-T T loop is located oppositely to the QRS loop

The location of the loop markedly to the left and posteriorly corresponds to the pronounced left axis deviation seen in the electrocardiogram with the shift of the transitional zone to the left Both the loop and the complex of the RS-T T phase are oppositely directed to the loop and the complex of the QRS phase The rapid inscription of the loop corresponds to the normal or moderately increased QRS duration

The distinction between the patterns seen in marked left ventricular hypertrophy and those found in left bundle branch block may perhaps be possible with the aid of the vectorcardiogram but this problem still requires further investigation

In *right ventricular hypertrophy* the spatial QRS loop is very characteristic and permits the diagnosis better than does the electrocardiogram (Fig 290) Most of the loop lies to the right and anteriorly The initial segment to the right and anteriorly is similar to the initial segment of the normal loop but then after it moves to the left for a short distance it proceeds anteriorly and to the right

though it is much smaller in size. In *young* patients it tends to lie posteriorly to the left and inferiorly, while in the older *groups* it

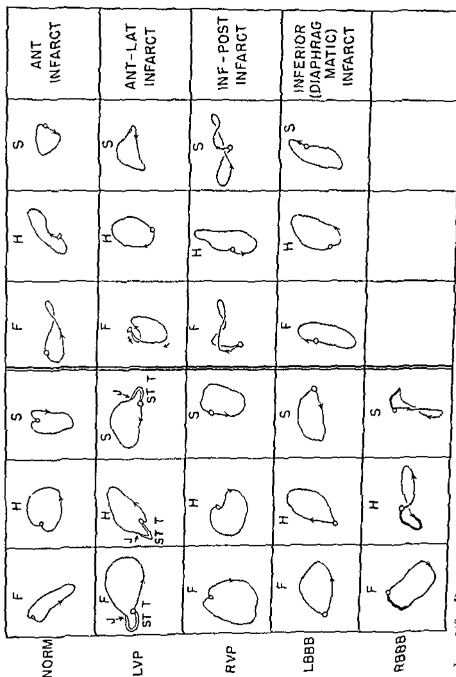


FIG 240 Diagrams of the QRS *loops in different conditions*. Only the QRS loops are drawn except in the case of left ventricular hypertrophy (LVH) where the RS-T T loops *are also indicated*. In this particular section J indicates the junction of QRS and RS-T T loops. J is not at the isoelectric (zero) spot. This corresponds to the marked deviation of the *junction J* in the electrocardiogram in this condition. In the case of right bundle branch block the heavy line indicates the terminal segment which is inscribed slowly. F, H and S indicate the frontal, horizontal and sagittal loops respectively.

lies more anteriorly and to the right of the QRS loop. Often the inscription is very irregular and the centrifugal limb is inscribed

At present it appears impossible to recognize abnormal right ventricular hypertrophy in the newborn whether with the electrocardiogram or the vectorcardiogram if only a single record is available. It is necessary to have serial records over the course of several weeks or months. A pattern of increasing right sided preponderance indicates that an abnormal right ventricular hypertrophy is present or developing. Normally the degree of right sided hypertrophy shows a decided tendency to decrease progressively (Schaffer).

The spatial loop in *right bundle branch block* characteristically shows a terminal segment to the right and anteriorly which is inscribed slowly and irregularly. Frequently the centripetal limb is deviated anteriorly. Otherwise the main loop is similar to the normal. The RS-T T loop is located opposite to the terminal appendage. The latter corresponds to the late broad S wave seen in Lead I and the left sided chest leads; it also corresponds to the late or second R (R' wave) in right sided leads. The tendency for the centripetal limb to deviate anteriorly corresponds to the positivity of the wave which lies between the two R waves in V_1.

The main part of the QRS spatial loop in right bundle branch block is similar to the normal. This is true especially for the initial segment which proceeds to the right and anteriorly and the centrifugal limb which proceeds to the left. Accordingly it becomes possible to recognize the presence of abnormal left ventricular hypertrophy or myocardial infarction in spite of right bundle branch block. In left hypertrophy the main loop tends to lie superiorly. The expected posterior deviation is neutralized by the anterior deviation inherent in this disturbance. In myocardial infarction the characteristic deformation of the early part of the QRS loop (corresponding to the abnormal Q wave) is also clearly evident in spite of the bundle branch block. However when right bundle branch block is present one should be very careful about making the diagnosis of myocardial infarction on the basis of abnormalities of the QRS loop alone. Abnormal conduction occasionally can cause a deformation similar to that seen in infarction.

In the typical case of *left bundle branch block* the QRS spatial loop begins with an initial deflection that proceeds to the left and posteriorly. The centrifugal limb likewise pursues the same path. The centripetal limb lies to the left of and anteriorly to the centrifugal limb. The entire loop lies superiorly and posteriorly. The frontal loop is always inscribed counterclockwise and is located superiorly and to the left (Fig 290). The horizontal loop is always inscribed clockwise and lies posteriorly to the left. The sagittal

The inscription is smooth rapid and without appreciable delay The frontal loop is always inscribed clockwise and is located mostly to the right The horizontal loop is always clockwise and is located anteriorly and to the right These characteristics of the horizontal loop permit an easy and reliable diagnosis of right ventricular hypertrophy No other condition gives the same type of horizontal loop The sagittal loop usually is inscribed counterclockwise and lies anteriorly for the most part The more pronounced the right hypertrophy is the more the spatial QRS loop tends to lie to the right, anteriorly and superiorly, under the same circumstances the RS–T, T loop tends to show more decided dislocation opposite to it

The correlation of the loops with the electrocardiogram is a very instructive process for it shows how the electrocardiogram may be misinterpreted As Fig 290 shows, the horizontal loop will project on the chest leads in such a way as to induce a double R complex in the right sided leads and a broad S wave in the left sided chest leads Thus, the electrocardiogram has the pattern of right bundle branch block Since however the width of the QRS is less than 0 12 second this has been called incomplete bundle branch block The loop indicates however that this pattern can also be due to a straightforward right ventricular hypertrophy without any evidence of a conduction disturbance Actually it has been impossible to decide from the electrocardiogram in such cases whether hypertrophy or block is present even if one is cognizant of the possibilities Under these circumstances the vectorcardiogram appears to be indispensable for an accurate diagnosis

In the younger age groups the electrocardiogram often displays the pattern of right ventricular hypertrophy In such cases, with the exception noted below the vectorcardiogram may show conclusively that the condition present is juvenile normal left preponderance namely a normal spatial loop similar to that in the adult except for a location somewhat more anteriorly and to the right In particular the horizontal loop is inscribed counterclockwise

The important exception to the above statement is the neonatal heart In the newborn the electrocardiogram is typical of right ventricular hypertrophy the same is true of the vectorcardiogram The loop is located to the right and anteriorly both the frontal and the horizontal loops are inscribed clockwise Furthermore the location of the loop superiorly to the right and anteriorly is often the same as is seen in older subjects with marked right hypertrophy (Schaffer)

left bundle branch block the septum is activated abnormally from the right to the left Accordingly the initial segment of the loop proceeds posteriorly and to the left

Vectorcardiography is well suited for the study of changes caused by *myocardial infarction* When an area of the ventricular myocardium is injured to the extent that it is no longer activated then its *normal contribution* to the formation of the QRS loop will be missing (Fig 290) Every instantaneous vector is the resultant of a multitude of smaller component *vectors many* of which are mutually antagonistic as has been stressed repeatedly in this book Inactivation of the myocardium which contributes vectors pointing anteriorly will permit the vectors which point posteriorly to dominate The resultant instantaneous vector will point away from the injured region

The normal direction of the initial segment during septal activation is anteriorly and to the right Should the anterior part of the *septum be infarcted then the corresponding part of the loop* (initial segment) will be directed posteriorly either to the left or to the right Deviations of the rest of the loop will depend on injury of the rest of the left ventricular myocardium Failure of the anterior wall to be activated will shift the spatial loop posteriorly This will be visible in the horizontal and sagittal loops but not of course in the frontal loop The latter plane does not have antero posterior dimension Failure of the lateral wall to be activated causes the spatial loop to deviate to the right this will be visible in the frontal and horizontal loops but not in the sagittal one Failure of the inferior wall to be activated *causes the spatial loop to deviate* upward This will be visible in the frontal and sagittal loops but not in the horizontal Failure of the posterior wall to be activated will cause the spatial loop to deviate anteriorly this will be seen in the sagittal and horizontal loops but not in the frontal one The distinction between posterior and *inferior location of injury* is of importance In the electrocardiogram the Q_2 Q_3 and QaVF pattern was often called posterior wall infarction Now with the aid of the vector loops it is clear that evidence of this pattern is more suggestive of an inferior wall infarction while the term *posterior* wall infarction should be reserved for those cases where the loop is deviated anteriorly In the electrocardiogram the latter type leads to abnormally high R waves in lead V_1 and V

The RS-T T loop and the part representing the PS-T segment (initial part of the RS-T T loop) show deviations in [illegible] relation to the infarction in the same *manner as the* [illegible]

loop is always inscribed clockwise and is located posteriorly and superiorly. There is always a delay in the inscription, usually in the middle of the loop. Usually the loop is smooth. The significance of gross irregularities is unknown.

The differential diagnosis between marked left ventricular hypertrophy and left bundle branch block is not always possible even with the aid of the vectorcardiography. It is still not known whether all cases showing the above mentioned type of loop actually have left bundle branch block, or on the other hand whether all cases of left bundle branch block show that loop irrespective of complications.

The RS–T loop in left bundle branch block is located opposite to the QRS loop. The junction between the two loops is away from the isoelectric point.

The marked superior and posterior location of the loop corresponds to the large S wave in Leads II and III, aVF and the chest leads as far left as V_4 and V_5. The marked deviation of the RS–T segment or J depends upon the same factors that are responsible for the QRS and RS–T T loops being not closed entities separated by an isoelectric line. The delayed inscription of the middle of the spatial loop corresponds to the notching and slurring at the peak of the R wave in left sided leads.

The influence of complicating factors on the spatial loop in left bundle branch block has not been determined. There has been little if any concrete evidence to suggest that it will be possible to recognize signs of myocardial infarction in such cases any better than it is possible with the electrocardiogram.

It has been pointed out that in right bundle branch block the QRS loop is shifted anteriorly while in left bundle branch block it is shifted posteriorly. Where the normally activated ventricle meets the delayed ventricle some activation will occur in an abnormal manner by way of the common myocardium. In the case of right bundle branch block this abnormal activation will proceed from the left (posterior) ventricle to the right (anterior) ventricle. In the case of left bundle branch block it will proceed from the right to the left ventricle in an antero posterior direction. This could account for the observed shifts of the loops (Schaffer).

The manner of septal activation as conceived at present could adequately account for the manner in which the spatial loops begin. In right bundle branch block the septum is activated normally from the left (posterior) side to the right (anterior) side. The initial segment of the loop is normal, namely to the right anteriorly. In

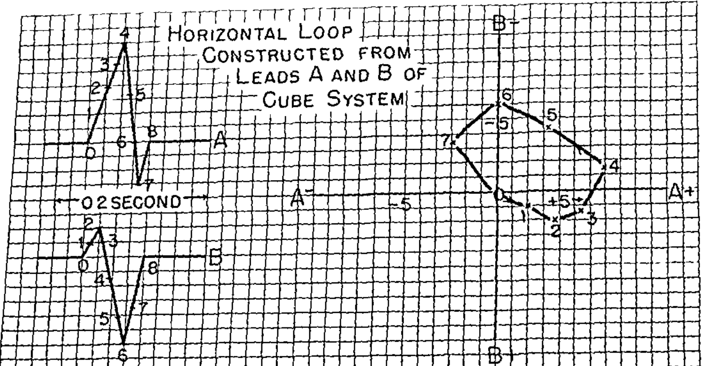


FIG. 91. *Diagram* illustrating the method for constructing a loop from two simultaneously inscribed leads A and B in leads inscribed according to the cube system. A is the horizontal, B the sagittal lead. They have been recorded on a two-channel electrocardiograph simultaneously. Points 1 through 8 indicate synchronous points calculated every 0.01 second apart. Plotting the synchronous deflections on the graph gives the horizontal loop. The method of graphing depends on the manner in which the figure is oriented. The latter is in accord with the vectorcardiographic method described previously. According to this method an upright deflection in A indicates a position to the left, while a positive deflection in B indicates a position of the point below (anteriorly). Lead A serves as the x axis, Lead B as the y axis. Successive points are joined to form a loop. Note that Lead B consists of a posterior and anterior electrode with the anterior electrode being the positive one. The cross section of the thorax is so oriented that anterior is located down; consequently an upright deflection in B means that the point in the graph is down. Therefore the positive side of the axis y is located down, the negative side up.

complexes in the electrocardiograms In the early phases of an infarction the first part of the RS-T T loop corresponding to the RS-T segment, will deviate toward the site of the injury later this type of deviation, provoked by the current of injury disappears and the entire RS-T T loop tends to deviate away from the injury

In comparison with the electrocardiogram, the extent of clinical and autopsy experience with the vector loops in the diagnosis of myocardial infarction is limited Therefore the new method should be applied with caution and the interpretations should be confirmed by the electrocardiogram With more experience it is possible that analysis of the loops will enable a more accurate and reliable diagnosis of infarction than heretofore possible This seems improbable in the small infarctions in which the QRS complexes show little or no changes

VECTOR ANALYSIS OF THE ELECTROCARDIOGRAM

The cathode ray oscilloscope picks up, as we saw two leads simultaneously and records them as a series of instantaneous vectors namely the vector loop If these two leads are registered simultaneously on a multi channel electrocardiograph by a graph one can derive the same instantaneous vectors and loop Clinically this method is impracticable since it is time consuming However a brief description of the method is in order so that it can be utilized in special cases It can serve as a substitute for a vector cardiographic apparatus

The same principles of lead systems are used as for direct vector cardiography To obtain the loop for each of the three planes one employs the same combination of leads described previously for example for the horizontal plane one records a lead with a horizontal axis and one with a sagittal axis simultaneously Registration is done at high speed if possible (5 to 10 cm per second) on a multi channel electrocardiograph One lead serves as the x axis the other as the y axis of a conventional co ordinate system for graphing Every 0 005 or 0 0025 second the synchronous deflection in each lead is plotted The plotted points which represent the positive tip of instantaneous vectors are connected chronologically to develop the loop The illustration in Fig 291 indicates the manner in which this is done Having constructed the frontal horizontal and sagittal loops the spatial loop can be reconstructed with a wire

However the routine electrocardiogram can be analysed and interpreted in terms of vectors quickly and simply by mental geometry' The value of this procedure has been discussed in

of the complexes. Thus the mean vector of the P, QRS and RS-T complexes is determined; then if desired one can determine the mean vector of the first part of the QRS complex of the RS-T segment or the Ta wave, etc. For purposes of discussion only the mean vector of the QRS complex will be used.

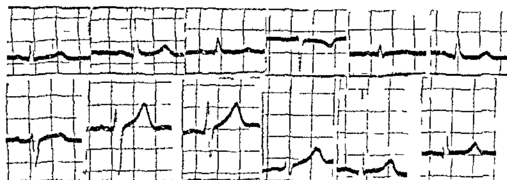
From the limb leads one determines the frontal aspect of the mean spatial vector. Inspection of Fig. 292 shows that the QRS complex is almost transitional in type in Lead aVL. (It shows slightly more positivity than negativity.) This indicates that the mean QRS vector is almost perpendicular to the axis of Lead aVL. Practically always at least one limb lead will be found whose complex is transitional or nearly so. The size of the frontal vector (which is of minor importance) is estimated roughly by noting the size of the QRS complex in the lead with the largest deflection (Lead II). The mean QRS vector will tend to parallel to the axis of this lead. The arrowhead is placed on the vector so that it projects positively on the axes of those leads whose QRS complex is positive and *vice versa*.

It remains now to determine the sagittal inclination of the mean QRS vector. This is accomplished by examining the chest leads. The axes of these are shown again in Fig. 292. The procedure is similar to that discussed in the preceding paragraph. The QRS complex in V_2 shows a transitional pattern. Therefore the QRS vector is perpendicular to the axis of V_2. The arrowhead is placed on the left end of the vector. According to the diagram the vector points a little posteriorly. The size of the vector in this dimension cannot be estimated because the distances between the electrodes and the heart vary so much that no approximation is possible.

The practical application of this method, especially the determination of the sagittal component, is not without its difficulties. However they can be surmounted if one will take the trouble to visualize the problem spatially. This is easy with the aid of a simple model.

An X-ray film of the chest should be rolled to form a cylinder and the approximate locations of the chest leads in positions 1 to 6 be marked. Now a long pencil is passed perpendicularly through the centre of a circle of stiff paper so that the pencil is bisected. The pencil represents the vector, the paper circle the transitional plane; all points on the latter are equidistant from the tips of the pencil. The point of the pencil represents the positive tip. Then place the model vector inside the rolled up film (the model of the chest) so that the centre of the pencil lies in the assumed centre of

detail by Grant and his collaborators In spite of the fact that the results are only crude approximations of the vector loops the clinical value of this method cannot be denied Actually it is better not to derive the loops at all because the method is too inaccurate It is much better to derive only the mean vector of different complexes or parts of complexes Hereby one avoids the pseudo accuracy which in turn leads to a false sense of diagnostic acumen



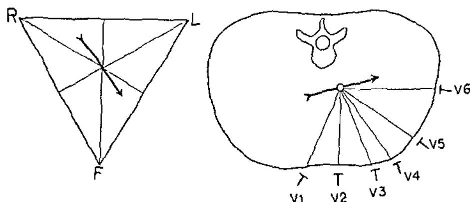


FIG 292 Method for determining the spatial mean vector of a complex In this case the mean QRS spatial vector will be found from the usual 12 lead electrocardiogram which is shown above

One needs a diagram of the axes of the routine leads plus the routine electrocardiogram The latter should include the six limb leads and the usual six chest leads Later it will be shown that on occasion certain other chest leads will be desirable

The diagram of the axes of the limb leads is shown in Fig 292 After a little practice one can remember the diagram sufficiently to apply the method mentally At first however it is advisable to make sketches The object is to determine the spatial *mean* vector of the different complexes and also the mean vector of certain parts

zone In general the principle of determining the sagittal inclination of a vector is to take a series of leads along a path more or less parallel to the vector so that one part of the series shows the positive side the other the negative side with the transitional position clearly defined The vector will be perpendicular to the axis of the transitional lead

This method of vector analysis has some advantages It affords a more logical explanation of the chest leads Vector analysis and vectorcardiography were helpful in showing that for all practical purposes the chest leads are not semi direct leads but distant leads like the limb leads which register the integral heart vector This method of interpretation avoids the many difficulties when the deflections in Lead V_1 were correlated with the activity of the right ventricle and those in V_5 with the left ventricle

This method clarifies the distribution of the complexes on the chest it shows how all leads outside or inside the body (even intra cardiac leads) can be logically related to the heart vector

Another advantage of this method is that it permits an interpretation of every deflection of the electrocardiogram in a clearer way It shows that whenever an activation front approaches the exploring electrode (or the positive electrode) of a lead the deflection is positive When the activation front recedes from this lead the deflection is negative When the recovery front approaches the electrode the deflection is negative when the recovery front recedes from this lead the deflection is positive

It is not permissible to construct the vector loops by the method of vector analysis described in this section if a single channel electrocardiograph alone is used because the single waves of the QRS complex are not formed simultaneously Another limitation is the absence of significance for the mean vector when the loop is circular The thinner the loop and the more elongated it is the more accurately the mean vector describes its spatial characteristics and *vice versa* The normal loops are particularly in the frontal plane elongated and thin ellipses

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the heart Where the plane intersects the chest wall, the transitional zone is defined An electrode anywhere on this zone will be equidistant from the tips of the vector and will record a transitional complex An electrode anywhere to one side of the zone will register a positive complex and one anywhere on the other side a negative *complex (see Fig 278)* Note how the transitional zone shifts with movement of the vector If the pencil points directly to the left and is not inclined anteriorly or posteriorly the transitional zone *appears at or near V_2 All leads from the right of V_2 will register* a negative complex , all leads to the left, a positive complex This will hold even for leads above and below the usual level of electrode placement (4th and 5th intercostal spaces) A shift of the pencil so that it points anteriorly results in a shift of the zone to the right If the zone is more right than V_1 then all six common chest leads will register a positive deflection and none will show a transitional one In order to find a lead with a transitional deflection one must now take a lead from the right anterior chest (V_3R or V_4R etc) or from the left back (V_7 V_8) If now the pencil is shifted to point *more posteriorly the transitional zone may shift to V_4 if the pencil* points directly posteriorly the transitional zone may be at V_6 The inclination to the front or back can usually be determined if one has a series of chest leads that cross the transitional zone in an orderly manner provided the vector is more or less horizontal The situation is not so clear when the vector is vertical so that it points up or down In this case the pencil represents in the model the mean vector of a perpendicular heart It will be noted that the transitional zone intersects the usual chest leads in a complicated fashion which changes markedly with a slight change of the paper disk with the pencil Depending on where the heart is assumed to be located the predicted complex can either be positive or negative Obviously neither the position of the chest leads nor the position *of the axes is that exact If the pencil points only slightly anteriorly* the electrode of V_1 will be on the positive side of the vector s electrical field , its QRS complex is definitely positive Lead V_5 may be on the transitional zone or close to it and may show a RS complex Lead V_6 may be on the negative side and show a rS complex This is the classical pattern of right ventricular hypertrophy It will be noted how much the pattern can be varied if the pencil *or the position of the electrodes* is shifted only a few millimetres

The sagittal inclination of a vector can be more reliably ascertained by recording a series of leads from above downward (œsophageal leads *for instance)* so that they cross the transitional

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